ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

INCORPORATING THE INDEX OF OTOLARYNGOLOGY.

VOL. XXIX. DECEMBER, 1920. No. 4.

LXXII.

LOCAL AMYLOID WITH SPECIAL REFERENCE TO SOCALLED AMYLOID TUMORS OF THE TONGUE.*

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MINNEAPOLIS.

Amyloidosis, or generalized amyloid deposits, usually appearing in the liver, spleen and kidneys and also in other tissues, are well recognized in medical literature. Although now found in a markedly lessened number of cases as compared with the days before early and rational surgery became a custom, the condition is by no means a rare one.

The ease with which amyloid in these cases may be recognized, both macroscopically and microscopically, its specific physical and chemical properties and its almost constant "locus" in the subendothelial spaces of blood sinuses and capillaries and in the walls of medium sized and small arteries, all together present such a classical picture that, aside from

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questions in regard to pathogenesis, little remains to be added to our knowledge on this subject.

Local amyloid deposits, on the other hand, are so infrequently recognized or described, and present so many modifications from the lesions seen in general amyloidosis that the study and report of instances of their occurrence are still fully justified.

LOCAL AMYLOID OF THE EYELIDS AND RELATED STRUCTURES.

Some of the earliest references to local amyloid disease are found in the ophthalmologic journals and deal with amyloid deposits in the conjunctiva, cornea, and other structures of the eye. The first reported studies of this condition seem to have originated from the University of Dorpat (now Yuryev), which was the center of a population heavily infected with trachoma. Not all of the literature has been found available, but the references which could be consulted were fairly representative, and the cases which are abstracted below reveal most of the noteworthy features presented by amyloid changes in the lids, cornea and scleræ. The cases are arranged chronologically:

1. Kubly. Adenomatous growth in conjunctiva of left lid with hyaline changes. Hyaline also seen in right lower lid and amyloid in conjunctiva of right upper lid.

2. Beselin. Masses of amyloid in superficial layers of cornea.

3. V. Oettingen. Male, age 51. Tumorlike thickening of left lower lid showing amyloid. Left upper lid and both lids of right eye showing trachoma.

4. V. Oettingen. Female, age 22. Right and left upper lids thickened and amyloid affecting smaller vessel walls identified.

5. Kyber. Adult. Tumor removed from inner angle of eyelids, diagnosed clinically as squamous cell carcinoma. Microscopically no epithelial cells found, only necrosis and amyloid degeneration of small vessels.

6. Saemisch. Male, age 33. Swelling of left lower lid. Trachoma of left upper and both right lids. Disease had lasted two years, following a cold. Operation on left lower lid

only. Chronic perichondritis of cartilage with amyloid changes in the affected cartilage.

7. Saemisch. Male, age 45. Findings similar to above in right eye. Both patients otherwise well.

8. v. Becker. Tumorlike thickening on tarsal portion of lower lid with amyloid deposits. Trachoma of upper lid.

9. Reymond. Amyloid tumor size of almond in scleral tissues of one eye only. No involvement of lids.

10. v. Hippel. Male, age 29. Swelling for two and a half years affecting both lids of right eye near outer angle and also left upper lid. Masses removed from all three lids and showed amyloid in clumps and also involving vessels, with foci of calcification and ossification. Regarded amyloid as a regressive metamorphosis of tissues of lids probably accompanying trachoma.

11. Leber. Male, age 22. Swelling of left upper lid for six years following operation for ptosis of lid. Other upper lid also thickened. Involvement of scleral tissues in both eyes. Patient otherwise well. Clinical diagnosis trachoma, but pieces removed showed amyloid of blood vessels and apparently in lymph channels. Thinks v. Oettingen's cases probably trachomatous, but doubts this diagnosis in his and Saemisch's cases. Regards amyloid as a degeneration following a new formation and sclerosis of connective tissue. Found cell structures suggesting giant cells.

12. Leber. Male, age 21. High grade diffuse trachoma and amyloid thickening of left upper lid. Piece removed revealed amyloid of blood vessels and in concretions. Giant cells present.

13. Leber. Saw similar findings in another old case of trachoma with very thickened lid.

14. Zwingmann. Reports a recurrence two years following operation. Regards amyloid as a regressive metamorphosis in diffuse or circumscribed granulomata, a sort of trachomatous overgrowth.

15. Narkiwicz. Found amyloid in conjunctiva near to a tumor of lid, a rhabdomyoma striocellulare, that also showed amyloid deposits.

16. Mandelstamn and Rogowitsch. Female, age 52. Both lids and scleræ around cornea of right eye thickened and

showed amyloid in walls of vessels and in lymph channels. Giant cells present. Trachoma in lids of left eye.

17. Bachelmann. Hyaline degeneration of conjunctival tissues with circumscribed amyloid deposits. Regarded the amyloid as secondary to the hyaline changes.

18. Raehlmann. Female, age 19. Bean sized amyloid tumor of upper lid. No reaction around it. No giant cells and no other evidence of disease. Believed amyloid followed hyaline changes.

19. Vossius. Male, age 35. Tumor showing amyloid in right upper lid of two years' duration. Measured 30 mm. long, 12 mm. in breadth and 6 mm. in thickest portion. Vessels involved and also found a tiny focus of ossification. Giant cells present. No evidence of hyaline.

20. Vossius. Female, age 19. Amyloid tumor of right upper lid, 22 mm. in length, 9 mm. in breath and 4 mm. in thickest portion. Amyloid resembled preceding case and in addition involved basement membrane of Krause's glands. Foci of calcification.

21. Krüdener. Collected six cases from Dorpat and two from Petrograd of amyloid tumors of eyelids. Vessels always involved. Thought amyloid followed hyaline changes.

22. v. Hippel. Enucleation of eyeball following traumatic cataract and iridocyclitis with leukoma adhaerens. Amyloid present in anterior layers of thickened cornea.

23. v. Hippel. Irregular thickening of cornea with clumps of amyloid in affected portions. Believes amyloid in both cases was derived from red blood cells.

24. v. Hippel. Male, 24. Cloudy cornea of left eye with blindness. Amyloid masses in thickened cornea.

25. Naito. Male, age 37. Left eye enucleated because of blindness following inflammation in infancy. Amyloid changes in obliterated vessels of retina and in connective tissues posterior to retina with bone formation. Regards the amyloid bodies as corpora amylacea deposited in optic nerve preceding hyaline changes.

26. Schieck. Male, age 19. Tumor of scleral conjunctiva extending from caruncle to limbus with later involvement of lower lid. Excised mass showed amyloid deposits and amy-

loid in vessel walls. Giant cells present; also infiltration of lymphocytes and plasma cells. Various intermediate changes between amyloid and hyaline.

Of 14 of the above cases where sex and age were given, 10 were males and 4 females. The ages ranged from 19 to 52, with the majority (8) of these occurring in persons under 30. In 19 cases the lids were involved and only one of these (No. 14) was a real tumor. The cornea showed changes in four cases and the scleral conjunctiva in three instances. In one (No. 24) the retina and adjacent tissues were attacked. Amyloid was noted in the vessel walls of 11 cases, giant cells in 5, hyaline changes in 5, ossification in 3 and calcification in 2. Two authors concluded that the deposits could be seen in lymph vessels.

While one must admit that in all of the above cases amyloid could not be shown to be absent in other tissues of the body, the conclusion seems nevertheless justified that inflammatory conditions of the lids, cornea, sclera and other portions of the eye may show secondary and strictly local deposits of amyloid.

The question of the relation of these deposits to trachoma has provoked much discussion and contrary opinions. There seems to be little doubt that many cases of trachoma are complicated by amyloid infiltrations, but similarly there are numerous examples of amyloid appearing in or about the structures of the eye without the slightest evidence of any recognizable preceding disease. The extreme grade of chronic inflammation often encountered in severe trachoma, associated with hyaline changes in the dense connective tissue of the tarsal plates, may be reasonably interpreted as one set of favorable or predisposing conditions for the appearance of local amyloid.

This lesion would seem to deserve more attention than it has heretofore received, especially from American and English ophthalmologists.

LOCAL AMYLOID IN THE RESPIRATORY TRACT.

Of all the regions of the body where local deposits of amyloid have been described, those found in the respiratory tract have received perhaps the most careful attention from pathologists and clinicians.

The respiratory tract has long been recognized as being the seat of what we must, from the present state of our knowledge, regard as lesions closely related to amyloid formation. Early and pathologic ossification of the laryngeal cartilages, ossification and calcification of the tracheal and bronchial rings, socalled metastatic deposits of lime salts in the tissues of the lungs, corpora amylacea in the alveoli, and the fairly frequent occurrence of bone and cartilage containing tumors of any portion of the tract, as well as the occurrence of amyloid, are all conditions which may at some future time be found to be associated in whole or in part with similar fundamental alterations in metabolism. The cases which are reviewed below represent the essential data available and are arranged in the chronologic order of their publication:

1. Lesser. Female, age 78. Death from aortic and mitral insufficiency. Large bone and cartilage containing tumor showing amyloid degeneration and measuring 15 by 5.5 by 2.5 cm., in lower lobe of left lung. Near mass other smaller similar nodules. Syphilitic scars in liver. Amyloid also found in spleen.

2. Balser. Male, age 66. Death from slowly increasing stenosis of bronchi due to development of numerous irregular gelatinous and colloid masses on inner surface of both bronchi and trachea, which were changed into hard inelastic tubes. These masses showed cartilage and bone formation with amyloid change. The amyloid also involved blood vessels of the submucosa as well as the basement membranes of the glands.

3. Hooper. Male, age 33. Operative removal from larynx following hoarseness, of a smooth, globular, slightly lobulated, pedunculated tumor 12 mm. in diameter and attached to anterior superior surface of larynx, involving left vocal cord and portion of ventricle. Peduncle short and 3 or 4 mm. in diameter. No recurrence after 10 months. Microscopic diagnosis, telangiectatic myxofibroma with hyaline and extensive amyloid degeneration. Amyloid also seen in blood vessels.

4. Schranck. Female, age 35. Operative removal of cherry sized tumor from anterior wall of larynx. Tumor

showed amyloid with transition stages from hyaline. Recurrence. Syphilis given as etiologic factor.

5. Lubarsch. Female, age 75. Nodules found in lung, composed largely of hyaline connective tissue with some amyloid deposits. Extensive review of relation of hyaline and amyloid degeneration.

6. Burow-Neumann. Male, age 66. Had suffered from dyspnea for 15 years. Seven years before death had two tumors removed from larynx, and from that time up to death from bronchitis lived with cannula in tracheotomy wound. At autopsy three tumors found in larynx obstructing the lumen. These were composed of amyloid masses arranged in concentric layers, with intervening portions of connective tissue. Process regarded as amyloid degeneration of fibromata, inasmuch as the two earlier tumors were called fibromata.

7. v. Schrötter. Female, age 57. Operative removal of tumor from left arytenoid cartilage. Amyloid in walls of tumor arteries. Believed to be a fibroma. Foci of calcification noted. No evidence of syphilis.

8. Glockner. Male, age 76. Death from pyelonephritis. Excrescencelike hard tumors above cricoid, as well as in mucosa of trachea and large bronchi. Microscopically showed to be ecchondroses, with extensive amyloid deposits in bands and clumps. Basement membranes of mucous glands and walls of small arteries involved. Ossified and calcified foci present. Large giant cells often flanked amyloid masses, between which was granulation tissue. Hyaline changes in connective tissue with transition stages to amyloid. Primary tumors regarded as multiple ecchondroses and exostoses, the amyloid representing a metaplasia of the connective tissue trabeculæ (arising from perichondrium) passing through cartilage and bone tissue formation as a result of chronic inflammation. No chronic bronchitis. Lymph nodes and other organs free. No evidences of syphilis.

9. Manasse. Male, age 63. Death from perforated duodenal ulcer. In larynx a large flat and several cherry pit sized tumors showing hyaline cartilage, amyloid and giant cells. Amyloid interpreted as occupying lymph channels. Blood

vessels involved. No evidence of primary connective tissue tumor.

10. Manasse. Male, age 50. Operative removal of tumors from gums, both tonsils and left arytenoepiglottic ligament. Central portions showed cells of sarcomatous characters with amyloid deposited as a network. Giant cells present and believed to be derived from lymph vessel endothelium. Amyloid change in blood vessels. Lymph channels apparently invaded. Original tumors regarded as connective tissue in origin. No recurrence after one year.

11. Mager. Male, age 60. Operative removal of hard projecting tumor attached to both vocal cords. Amyloid as well as islands of cartilage and bone found. Vessels, glands and connective tissue showed amyloid. Tumor believed to be a fibroma with degenerative changes leading to bone and cartilage and amyloid formation.

12. Couvoisier (also Johanni). Female, age 66. Death following tracheotomy because of sudden suffocation. Clinical diagnosis, carcinoma of larynx. At autopsy, on the posterior wall of larynx opposite cricoid cartilage was a firm projecting tumor, 4.8 cm. in height, 2.8 cm. in breadth and 1 cm. in thickness. The flattened upper surface was covered by small nodules. Tumor had a yellowish brown color. Microscopically, tumor contained cartilage and amyloid. The latter involved walls of blood vessels and mucous glands, forming basement membrane rings and in places completely obliterating the gland lumina. Lymph vessels supposed to be filled with amyloid and amyloid bodies believed to be found in the capillaries. Suggests derivation from red blood cells. Giant cells noted at periphery of amyloid masses. Derived these from endothelial cells. Transition stages found between hyaline changes in connective tissue and amyloid. Perineurium of nerves also involved. Regards chronic inflammation as cause of amyloid deposits in the interstitial tissues. Lymphoid and plasma cells in mucosa and bone formation in cricoid cartilage. No amyloid in kidney, spleen or intestine.

13. Gerling and Hüter. Male, age 63. Hoarse for four years. Tracheotomy because of dyspnea. Two symmetrical tumors on posterior wall of larynx, beneath glottis, composed of amyloid masses, with a vascular, cellular intervening tis-

sue. Walls of arteries involved as well as basement membranes of mucous glands. In granulation tissue of submucosa amyloid extended to epithelium. Amyloid also supposed to be in lymph channels.

14. Herxheimer. Male, age 65, suffered ten years from dyspnea and hoarseness. Death from empyema. At autopsy, besides a large abscess in right lung extending to pleura, were found numerous firm nodules up to 1.5 cm. in diameter in right lower lobe and a few in left lung. These tumors were diagnosed as gummata but found to be amyloid. The larynx showed two large symmetrical tumor masses extending from epiglottis to cricoid, with destruction of vocal cords and narrowing of lumen, but no ulceration of mucosa. Also there was a similar tumor mass in trachea. The masses extended to the cartilages but were not attached to them. Microscopically, tumors composed of amyloid masses with foreign body giant cells, foci of calcification and amyloid in walls and lumina of mucous glands, walls of blood vessels and alveolar septa of lung and elastic tissue of pleura. Believes amyloid a primary process, though preceded in some portions by hyaline changes. Is certain amyloid found in lymph channels. Epithelium normal. No amyloid in other organs. No history or signs of syphilis. Careful comparative review of literature.

15. v. Werdt. Male, age 71. History of hoarseness and dyspnea for six years. Death followed shortly after operative removal of tumor on inner wall of arytenoepiglottic fold. At autopsy, in mucosa of trachea on membranous portion were four or five tumors averaging 4 to 5 mm. broad, 3 to 4 mm. high and 1 to 2 mm. in thickness. These reached to primary bronchi, where rows of smaller nodules extended to and into secondary bronchi on both sides. Appearances likened to stalactite formation. Bronchi of right upper lobe calcified and a bone hard tumor fills lumen of bronchus leading to this lobe. Bronchi to other lobes on right side free. On left side bronchi involved throughout. Microscopically, tumors composed of cartilage and bone in varying amounts with extensive amyloid formation. Mucous glands and walls of arteries involved. Giant cells and foci of calcification present. Amyloid believed present in lymph vessels. No hyaline changes noted. Corpora amylacea seen in lung alveoli. Diffuse inflammation

of mucosa of trachea but tumors purely local. Amyloid found in peritracheal lymph nodes but not in any other organs. Regards amyloid as local disturbance in nutrition caused by chronic inflammation.

16. Meyer. Male, age 57. Death from chronic nephritis and apoplexy. In lungs, which weighed 2,000 grams, were hard nodules, the largest (size of an apple) in lower lobe; another, size of small apple, in upper lobe, and numerous smaller, pea to bean sized nodules, throughout pulp. Tumors composed of cartilage and islands of bone with amyloid deposits. Giant cells present. No amyloid in liver, spleen or kidneys. Suggests chronic congestion of lungs as an etiologic factor.

Of these 16 cases, 11 were males and 5 females. The ages ranged from 33 to 78 (average 61). There were 11 cases showing involvement of larynx, three of trachea and bronchi and three of lung. In eight of the cases bone and cartilage were present, and three authors noted lime salts. Syphilis was present in two cases. Transitions from hyaline tissue were recorded six times, and in the same number giant cells were observed.

LOCAL AMYLOID OF THE TONGUE.

The localized presence of amyloid in the tissues of the base of the tongue presents perhaps the most obscure problem furnished by instances of this peculiar disease. A rather careful search of the literature reveals the following cases which are reviewed in chronological order:

1. Ziegler. Male, age 67. Three rounded or oval nodules in base of tongue, two on left side, one on right. Raised above surface but covered with intact epithelium. Masses of amyloid, walls of vessels involved. Larynx thickened on posterior wall and in region of cricoid. Two pedunculated polyps on false vocal cords, which showed amyloid change. Scars in liver diagnosed as syphilis. Traces of amyloid in scar tissue and in liver capillaries near scars.

2. Zahn. Male, age 38(?). At base of tongue on right side two rounded firm projecting tumors, one 13 mm., the other 19 by 12 by 15 mm. Central portions showed bone and cartilage with surrounding amyloid and hyaline changes. Amy-

loid also in rings around gland. Foci of lymphocytes. Author believed tumor originally an osteofibroma.

3. Kraus. Two amyloid nodules at base of tongue in midline.

4. Schmidt. Male, age 58. Death from emphysema of lung and bronchitis. At base of tongue on right side was a projecting tumor 18 by 15 mm. Inside of this, near midline, a smaller buried nodule, 8 by 5 mm. Amyloid in masses and involving basement membranes of glands. Calcification and small islands of bone in mid portions, also small rounded masses of hyaline cartilage. Walls of arteries involved. Concluded amyloid was in lymph vessels. Giant cells present. Lymphocytes present. Small mass of cartilage in right tonsil. No evidence of syphilis. No amyloid in other organs.

5. Schmidt. Female, age 60. Death from emphysema of lungs. At base of tongue on right side was a firm buried nodule 9 mm. in diameter. Resembled other case in every respect. Small center of bone. No signs of syphilis or amyloid in other organs. Believes all such tumors closely allied to bone and cartilage production.

6. Martucelli. Pedunculated tumor size of walnut at base of tongue.

7. Saltykow. Male, age 80. Death from apoplexy and tuberculous pneumonia. At base of tongue a pea sized abscess. To left of midline a cherry pit sized firm mass in submucosa showing amyloid in rounded masses with giant cells and involving glands and arteries. Small foci of calcification and bone formation. Review of literature.

8. Edens. Male, age 39. Death from accident. In base of tongue a slightly raised tumor 2.2 cm. by 1.2 cm., showing amyloid which enclosed glands by rings and contained lime salts but no cartilage or bone. Lymphocytes in neighboring tissue. Scar on prepuce and syphilitic aortitis. Regards amyloid as a primary chancre in tissues. Reviews literature. Believes amyloid due to circulatory disturbances and local inflammation.

9. Gross. Male, age 57. Operative removal (first exploratory for diagnosis; later whole tumor) hazelnut sized mass from base of tongue giving swelling under mucosa and extending nearly to epiglottis. Small abscesses near tumor

found at operation. Clinical diagnosis, carcinoma. Patient showed no recurrence at end of year. Tumor revealed masses of amyloid involving membrana propria of glands and walls of vessels. Intervening delicate connective tissue fibrils. No evidence of amyloid in lymph channels. Scattered groups of lymphocytes. No history or signs of syphilis. Patient suffered for years from hoarseness. This and local abscesses probably favored amyloid formation.

10. Henke. Cherry sized dense amyloid tumor at base of tongue, showing giant cells, cartilage and calcification.

11. Heller. Male, age 71. Death from chronic emphysema of lungs. Two projecting tumors at base of tongue, one on each side, the right size of walnut, the left size of cherry. Amyloid involving glands and blood vessels. Lymphocytes in region of amyloid. Foci of calcification and ossification. Masses of amyloid seen in supposed lymph channels. No evidence of syphilis or previous local tumor. Mentions observing amyloid change in stroma of carcinoma at base of tongue.

12. Schilder. Female, age 56. Death from pulmonary tuberculosis and bronchopneumonia. In base of tongue three small nodules ranging from size of hemp seed to that of a pea. Masses showed amyloid affecting mucous glands and muscles, fading into surrounding tissue. Stroma infiltrated with lymphocytes and plasma cells.

13. Schönhof. Male, age 62. Death from chronic tuberculosis of lungs. At base of tongue to right of foramen cecum was pea sized projecting, half rounded, dense, translucent tumor. Smaller similar nodules in mucosa directly in front of foramen cecum. On right margin of tongue opposite base were two pea sized waxlike tumor masses and on left side a somewhat smaller projecting nodule. On the right side of ventricle of larynx and involving the right false vocal cord was a small lobular tumor, and in the midline of the larynx as well as in the depths of recessus pyriformis were several flat wartlike nodules. All these tumors showed amyloid with giant cells affecting tunica propria of glands and striated muscles. Amyloid apparently in lymph channels. Surrounding stroma contained lymphocytes and plasma cells. No cartilage seen. Suggests tuberculosis with local inflammation as etiologic factors.

As with local amyloid in other locations, the male sex is predominantly attacked. Of the ten cases in which age and sex are given, eight were males and two females. The average age is 59 years, with extremes of 39 and 80. The late incidence of amyloid in the tongue and respiratory tract as compared with that in the eyelids is partially explainable by the fact that, as a rule, amyloid in the former location is only revealed at autopsy, while in the latter it is readily discernible from its early inception. Other and more obscure factors, however, must play a major rôle, as the average discrepancy in ages of the two sets of conditions is about 30 years.

In addition to the three sites for local amyloid, cases of which have been discussed above, scattered references to amyloid in other locations have been collected and will be classified and briefly reviewed.

LOCAL AMYLOID IN TUMORS.

1. Fitz. Female, 30(?). Lymphoma size of pigeon's egg in neck at angle of jaw of three years' duration. Greater portion amyloid. Some leucocytes.

2. Kruckmann. Male, age 61. Tumor of side of neck of sarcomatous nature showing hyaline and amyloid, bone and calcification, and giant cells.

3. Hildebrandt. Female, age 36. Round cell sarcoma of sternum containing corpora amylacea giving reactions for amyloid. Collected six cases of tumors with similar findings and two cases with corpora amylacea not giving reaction.

4. Lohrisch. Male, age 49. Mixed cell sarcoma of mediastinum containing corpora amylacea giving amyloid reaction. Discusses pathogenesis of these bodies.

5. Ophüls. Male. Case of syphilis showing multiple tumors of ribs, spinal column and sternum with amyloid and amylaceous bodies in tumors. Believes tumor syphilitic. Amyloid also in kidneys, heart and spleen.

6. Tschistowitsch and Akomow: Male, age 42. Death from pneumonia. Hard tumor, size of man's fist, in retroperitoneal region between folds of mesentery. Cut surface showed caseous appearing areas. Microscopic, thick network of fibers with lymphoid cells in between. Amyloid in stroma,

in walls of arteries and in lumina of lymph channels. Hyaline changes in connective tissue. Lymph and blood spaces dilated into angiomalike formations. Ulcer in small intestine in close relation to tumor. No evidence of syphilis. Case regarded as unique.

7. Edens. Female, age 66. Death from chronic nephritis. Amyloid mass size of a small apple on left sixth rib. Amyloid also in liver, spleen and kidneys.

8. Jaquet. Female, age 48. Lymphosarcomatous tumors in thyroid, lymph nodes of neck, liver, mediastinum and left axilla. Primary in mediastinum. Amyloid in mediastinal tumor in stroma and arteries; also nodes of neck. None in nodule in liver and none in pulp of liver, spleen or kidneys.

9. Hueter. Male, age 58. Case of chronic nephritis and multiple myelomata of ribs, sternum, left clavicle, humerus, scapula, around left shoulder and in ileum and jejunum. Tumor around shoulder and ulcerating tumors of small intestines contain amyloid. Refers to case of Askanazy.

LOCAL AMYLOID IN INTESTINES.

1. Schnützen (reference by Edens).

2. Neumann reports cases (reference by Askanazy).

3. Askanazy. Female, 58. Death from multiple myeloma and chronic nephritis. In jejunum on outer surface numerous nodules from almost invisible to size of pinhead, showing amyloid involving muscles and vessels. No amyloid in liver, spleen or kidneys.

LOCAL AMYLOID IN URINARY BLADDER.

1. Solomin. Female, age 73. Death from chronic nephritis. Posterior wall of bladder, dense homogeneous yellowish layer 8 cm. in diameter, with some ulceration and composed of amyloid. Giant cells present.

2. Luksch. Female, age 24. Death from pulmonary and laryngeal tuberculosis. In urinary bladder were protuberances of irregular size and yellowish color seated in muscular layers up to 2 cm. in diameter. Masses showed amyloid with foci of calcification. Regarded the tuberculosis as an etiologic factor.

LOCAL AMYLOID OF URETHRA.

1. Tilp. Male, 58. Death from lobar pneumonia. History of alcoholism. No history of gonorrhea. Amyloid mass in midpart of spongy portion measuring several centimeters in length and 2 to 3 mm. in height. No inflammation noted. No amyloid in liver or pancreas.

LOCAL AMYLOID OF HEART.

Beneke and Bönning. Male, old. Death from bronchitis. Marked atrophy of all organs. In heart and in vessels of lungs found amyloid. None in liver, spleen or kidneys.

LOCAL AMYLOID OF NOSE.

1. Grawitz. In the nose of a horse, age 7 years, several tumors from size of hazelnut to walnut which obstructed respiration. With a tube in trachea horse worked nine and one-half years. When killed nostrils completely filled with cauliflowerlike hard masses of amyloid involving vessels and basement membranes of glands. One wartlike amyloid tumor in trachea near artificial opening where there was marked chronic inflammation. Thinks case one of rhinoscleroma.

2. Rabe. Saw connective tissue tumors with amyloid on each side of nasal septum of a cat.

3. v. Joest. Amyloid deposits in nose of a horse: also amyloid in breast tumor of a female dog and in multiple tumors of domestic fowls, especially turkeys.

4. Wolf. Describes amyloid tumors in nose and trachea of a horse.

5. Roeger. Male, age 35. Tumor of nose with structure of a round cell sarcoma and showing hyaline and amyloid changes.

The case which forms the basis for this review was that of G. H., male, age 40, who was admitted to the university hospital five weeks before his death, presenting the typical signs and symptoms of toxic thyroidism. His history revealed, among other things, the occurrence of chancroid, buboes and gonorrhea at the age of 19 and a chancre at the age of 36. His blood Wassermann reaction was positive two years later. He was married at 24 and had one child, 11 years

old. His wife had one miscarriage. Evidences of toxic thyroidism had been present for the past four years, one operation three years ago giving him only partial relief. While in the hospital he showed marked weakness, a fine tremor of the fingers, exophthalmos, a pulse ranging around 120, and a prominent thyroid tumor which gave a thrill and bruit. Differential blood count showed the presence of 50 per cent lymphocytes on two different occasions with total leucocytes of 6,850 and 7,150 per cu. mm. The blood Wassermann reaction was positive on three different occasions, but two Wassermann tests of the spinal fluid were both negative. There was marked pyorrhea alveolaris.

Postmortem examination, performed sixteen hours after death, revealed the following conditions:

1. Hyperplasia of thyroid gland (weight, 195 grams).
2. Acute purulent tracheobronchitis with bronchopneumonia.
3. Syphilitic aortitis.
4. Hypertrophy and dilatation of heart with coronary sclerosis.
5. Old healed fibrous pleuritis.
6. Operative scar at base of neck.
7. Persistence of thymus (weight, 60 grams).
8. Pyorrhea alveolaris.
9. Acute and chronic tonsillitis.
10. Ossification of tracheal and bronchial cartilages.
11. Tumor mass at base of tongue.

The mass in the tongue represented almost an accidental finding. While demonstrating the inflammatory conditions of the tonsils and trachea a firm area was felt in the tongue and section revealed a well localized hard grayish white nodule about 1.5 cm. in diameter and lying deep in the muscle behind the circumvallate papillæ and well to the right of the midline. There was no appreciable swelling of the mucosa or tongue substance which might suggest the presence of the tumor. It was not encapsulated but firmly attached to the tissue of the muscle and surrounding mucous glands. Its cut surface was smooth and with its resistance to sectioning and whitish appearance suggested a chondroma. Portions were fixed in 10 per cent formol and Zenker's fluid.

Histologic examination shows a central area composed of a homogeneous, hyaline substance arranged in irregularly shaped bands and rounded masses which were partially fused, with occasional intervening clefts showing connective tissue nuclei and a few fibrils. With methyl violet and also with iodin this substance gives a staining reaction typical of amyloid. In the midportion are a few spicules of osseous formation, and surrounding this are occasional collections of basic staining granules suggesting calcium salts. At the periphery of the mass the amyloid invades the glands and surrounding muscle and fat tissue by irregularly projecting nodules which exhibit a gradual transition to normal appearing tissue without well defined or sharp limits. The mucous glands show changes which vary all the way from complete obliteration and replacement by rounded clumps of amyloid to structures in which only a narrow circle of amyloid has replaced the basement membrane. These "circles" are quite conspicuous, wherever the amyloid borders gland lobules. The epithelial cells lining such affected glands are visible in all but those in which the obliteration is complete, often showing as a small collection of atrophic cells in the center of a thickened amyloid ring. A few clumps of amyloid in the region of these glands are surrounded by a narrow but distinct zone of flattened cells suggesting a channel which the amyloid substance has invaded or in which it has progressed. Toward the striated muscles gradual changes are not so easily observed. At no point can be distinguished any hyaline changes in the muscle fibers themselves. The amyloid exists in the connective tissue trabeculæ, which have replaced the lost muscle and which in a few places show a true hyalinization not reacting to the tests for amyloid. No involvement of vessel walls was noted.

The epithelial lining of the tongue above the mass is normal, but the underlying connective tissue, including the unaffected mucous glands, shows a focal infiltration of lymphocytes and plasma cells and in places distinct increase of the fibrous connective tissue. Careful search in sections from the kidney, spleen, liver, lungs, bronchi, tonsils, thyroid, thymus, heart and aorta fails to show any evidenec of the presence of any substance even remotely suggesting amyloid.

DISCUSSION.

A review of this case shows that in a man, aged forty, who suffered from syphilis and toxic goiter, there was also found a small mass of amyloid about the size of a split pea, embedded in the substance of the right side of the base of the tongue behind the circumvallate papillæ. This mass contained bone and lime salts and was definitely invading the surrounding structures, attacking the mucous glands by first forming a ring at the site of the basement membrane and gradually replacing the entire gland. No transformation of striated muscle into amyloid was observed, but apparently the muscle substance was replaced by fibrous connective tissue which, in turn, became hyalinized and was then replaced by amyloid. That the hyaline was a precursor of amyloid change in these areas cannot be doubted. The amyloid clumps surrounded by a circle of flattened nuclei, which to so many authors has suggested a resemblance to lymph channel endothelium, is subject to another interpretation. This appearance was seen only in the region where mucous glands had been destroyed and the connective tissue stroma of these glands often retained its identity even when the gland structure itself had almost entirely disappeared. The encircling flattened nuclei may well represent, in every case in which they are described, not lymph vessel endothelium but surviving connective tissue stroma cells, so often appearing in the midst of amyloid masses seen in the liver, spleen and kidney, in cases of general amyloidosis. In respect to the genesis of such deposits, the case gives suggestive hints. In the first place, there is present ample evidence of local chronic inflammation, a phenomenon which has been interpreted by some authors as preceding, and by others as resulting from the presence of the amyloid tumor. The occurrence of local amyloid deposits following chronic trachoma, the constant association of general amyloidosis with chronic infections, as well as the absence of any real evidence of local irritation produced by accumulations of amyloid in the body, all point to the reasonableness of the conclusion that in this, as in other cases, the inflammation preceded the amyloid but afforded a basis for the changes preceding its deposit. Of further significance

is the almost constant association, as in the present case, of foci of ossification and calcification, as well as hyaline changes in the adjacent connective tissues with the amyloid masses. In the case under discussion this tendency to early changes in comparatively inert substances is emphasized by the discovery of ossification occurring in the tracheal and bronchial cartilages. The presence of a general chronic disease, such as syphilis, and, in addition, toxic goiter, furnishes sufficient basis for postulating a theory that local metabolic disturbances bring about a destruction of the more highly differentiated elements with some close relation between ossification and amyloid formation in the resulting process. The discovery by chemical analysis of chondroitin sulphuric acid in amyloid emphasizes the chemical resemblance of amyloid to hyaline cartilage, and even in those cases where this acid could not be found this resemblance is still suggested by the similar physical properties and staining reactions of the two substances.

A process, then, so closely allied to the formation of cartilage that true bone may be laid down in it and that results from a disordered metabolism produced by local inflammation plus generalized systemic intoxications may be a reason, if not a satisfactory explanation, for these strange formations. The occasional local deposits of amyloid in the mucous membranes of the respiratory tract in close association with cartilage forming connective tissues lends further support to this conception.

Why amyloid should be formed in the base of the tongue and always, when it does occur, in the same portion (posterior to the circumvallate papillæ) is not at all clear. The comparative frequency with which the follicular glands are subject to inflammatory processes, close association with the hyoid bone, developmental confusion of the tissues of that part of the tongue because of the thyroglossal duct, or the relative immobility of that portion and because of this fact and the peculiarities of its circulation, its exposure to chronic congestions of blood and, more especially, lymph circulation, all may be urged as reasons for its vulnerability to this form of degenerative lesion. The true explanation for this fact, as well as the comparatively infrequent occurrence of local

deposits of amyloid in the urinary bladder, intestines, lymph nodes and tumors must remain obscure until such time as we can fully understand the exact conditions under which this peculiar insoluble proteid amasses itself in the tissue spaces.

One further point deserves consideration. The frequent association of syphilis with the occurrence of both general and local amyloid deposits raises the question whether this substance may not be a direct local reaction to the syphilitic virus, a reaction closely allied to the usual gummatous type of necrosis. Perhaps local amyloid tumors are only modified gummata. The facts do not, however, favor such an assumption. Not only does amyloid occur in cases which are completely free from any clinical suspicion of syphilitic infection, but also these tumor deposits often fail to show any lesions which would suggest a syphilitic origin.

Finally, there seems little or no justification in this case or, indeed, in a large majority of the other cases, for postulating the previous formation of a fibroma or other tumor process. The histologic picture alone negatives this view and, while amyloid undoubtedly does occur in tumor formations, we must regard the tumor as an accidental locus and not in any sense a determining factor of the amyloid disease.

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LXXIII.

TRIFLES THAT COUNT FOR EFFICIENCY.*

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"Perfection is made of trifles; and perfection is no trifle," said Michael Angelo.

Not every trifler becomes a Michael Angelo; nor will any of us, unless, like the great master, he gives the greater part of his energies to the major aspects of his work, heeding but not unduly stressing these minor matters. When I try to give you what may be called "wrinkles," I hope they will not with you be merely "crows' feet," as you laugh at the elder member trying to teach you things you may have cut your medical teeth on. Our caustic friend, John Green, once said that he "had to search the dustiest archives of the specialty to find the new things Dr. Blank brought out in his communications." I have little doubt that most of the points I wish to make are well known to all of you: yet it is a fair question—"Do you sufficiently impress them on the attention of those whom you teach?" It is mainly because men of long experience in our specialty have praised some of them as new or ignored that I venture to bring them here forward.

In obtaining the histories of our cases, have we not commonly found that many of our patients' data are vague, contradictory or irrelevant and that we must correct them in the light of our actual findings? "I never had trouble with that ear," reported patients with perforated, scarred or pus crusted drumheads. Dr. Edward Woakes always looked first to the drumheads of his nose and throat cases to see what tales they might tell upon the upper respiratory tract; and it is good policy to reverse this and begin in our ear cases by examining the nares and fauces. Yet some would say that it takes too long thus to study the side issues—hence the importance of taking short cuts and learning to inspect the nasal passages

*Presented with twenty chart illustrations at the American Otological Society meeting, Boston, May 30.

fairly adequately by lifting the nose tip with the thumb, spreading the ala. Most men have the patient say "ah" to show the fauces: have them inspire noisily and one can commonly dispense with the tongue depressor, as with the speculum in the nose. We cannot always stop to explore the lungs, especially with bared chest, upon mere suspicion of tuberculosis; but we can detect readily, even by inspection, quite surely by auscultating through the clothing, and prolongation of the expiratory sound at the apices such as is usually an early, almost prodromal, sign of tubercular infiltration of that region.

In examining the auditory canal and drumhead—better that one first which is supposedly normal—we can often dispense with any specula if we rightly use traction—up and back in adults; down and back in young children—on the auricle and look through the sight hole of the mirror. In thus placing the eye in the center of the full cone of light from the head mirror, we secure better illumination for the whole drumhead as well as for the upper pole especially. This counts greatly in narrow canals, as in infancy, but most when using the pneumatic speculum, for with it much of our light goes to waste, reflected from the inner as well as the outer surface of the glass closing the speculum, and if this be a magnifying lens the loss counts four-fold. An older colleague used to teach that only a beginner needs this sight hole; so soon as he got out of baby clothes he turned his mirror up on his forehead and looked under it like a master. I have always believed that this explained why in 23 years he had seen but 20 cases of attic suppuration with Shrapnel perforation, of which I reported several series of 40 a year, as more correctly showing their frequency.

In thus drawing the ear straight we can at once notice any indurative stiffness of the canal and diagnosticate eczematous infiltration, even though all surface lesions are absent.

In our testing of the hearing, does any treatise advise that the tuning fork be always pointed towards the ear so that the sonorous vibrations of each tine shall reinforce, not neutralize, those from the other? Thus we get maximal audible, with minimal palpable vibrations, so that the patient is less likely to claim that he hears what he only feels. Would the Galton whistle be as lightly regarded if its opening were

always turned from the ear, its sounds elicited by successive pressures of its bulb without any intake of air, and its orifice held near the Darwinian tubercle at the upper margin of the helix where its whistle is readily heard to the limit, while its puffing is nearly inaudible? Scrutinize the whistle offered you as Galton's to make sure that it somewhat approximates the genuine, does not sound to or above the zero point, etc. Need we cumberously time with a stop watch the duration of bone conduction in Schwabach's test and the normally double sounding time for air conduction, when the Roosa Rinné test tells us in an instant whether the bone or the air conduction preponderates; and the Gardner-Brown test as quickly tells how much the bone conduction is above or below the normal? In case of tinnitus, must we scruplously cleanse the nares and pharynx and inflate by catheter or bag to test eustachian patulency? when by Politzer's test with the tuning fork vibrating before the nostrils the patient may at once tell us whether or not the tubes open in swallowing and pass the sound waves more fully to the tympanum of one or both sides.

When our tuning forks tell us that we are dealing with nerve deafness (combined or not with obstructive lesion), must we get a Wassermann report—perhaps sowing discord in families, since the public is quite alive to the significance of "blood tests"—when the evidence may be already staring us in the face? Not that the serrated tooth edges are Hutchinson's "notched teeth," as I have heard them designated; nor that the teeth of the second set need be notched to tell the story. The pegged teeth, with curved sides and narrow cutting edges, widely spaced, are almost as typical, though marking a more attenuated inheritance. I have never forgotten a boy with bad corneæ not improving under the care of a skilled oculist, who did not seem to have a mere phlyctenular keratitis, although it was not typically interstitial. He had some percussion dullness and prolonged expiration at the apices. In spite of perfect teeth, I asked of the physician who brought him as to the family history, any miscarriages or other luetic evidence, but learned only of a runaway father of a bad sort. At a later visit an elder sister brought him, a plendid six foot Juno of a trained nurse, whose teeth were

typically Hutchinson, justifying the antisyphilitic medication which was beginning to secure his improvement. At her next visit she had had all notching filed out of her teeth; but they showed still their spacing and pegging. In another case, a well developed girl of 15, the extreme deafness was ascribed to mumps and proved to be nerve deafness. Neither she nor a young cousin accompanying her could give any significant further history and, with well shaped teeth, I was about to accept the mumps as cause, when a faint violet injection around the corneæ caught my eye, the ophthalmoscope showed a vascularized interstitial keratitis and a closer look showed the teeth to be artificial. In a wholesome looking girl of 15 the upper incisors must have been pretty bad to require this replacement. She improved distinctly but not lastingly under antiluetic treatment. In many other cases where the taint is doubtless acquired and the teeth are normal and the hard palate is not suggestive, the velum shows brick red quadrate patches which Carl Seiler called attention to as positive evidence of syphilis. These are not marginal, as are most palatal injections, but symmetrically placed nearly in the middle of each side of the soft palate.

Of course it should be needless to refer to the typical specific facies, with scarred mouth angles, exaggerated nasolabial lines, saddle nose and specific tarsitis which may accompany and accentuate the keratitis. In examining the pharynx and larynx we have the good general rule, of which the instances outnumber the exceptions, that posterior lesions are tubercular; anterior, syphilitic. No small group of nerve deafness cases are merely neurasthenic and fleeting, and we note a scalloped, tooth marked tongue margin as indicating nux vomica for the flabby patient who has so puttylike and flabby a tongue.

As in the inspecting, so in the treatment of the nose and throat these simple measures are of great convenience. The thumb serves not only to reveal the anterior nares but, as a rest for the atomizer tip, relieves all chance of giving or receiving infection by its contact, makes injury through sneezing or other sudden forward movements of the patient unlikely; while the slight abrasion of the mucosa which the spray

itself can cause is obviated by the distance of the tip outside the nose. Our Philadelphia method (Walter Chrystie) of one-hand management of the ball atomizer makes this convenient at all times as well as at the bedside; and I rarely use the compressed air tank in spraying, considering a pressure of from five to ten pounds adequate and usually safer; and through the catheter it is satisfactorily used up in the eustachian tube, penetrating at least as far as by any other method. In this connection I would renew claims for S. D. Risley's method of using the ball tipped Politzer bag, which never jams in the catheter, yet gives good pressure without leakage. Any valve in the bag is needless, and medication of the air is conveniently secured by merely filling the bag in the mouth of the bottle of iodin, ether, etc.; and Lowenburg's admirable test whether chloroform vapor feels cold in the otosclerotic tympanum may aid us in that difficult diagnosis. Incidentally it may be said that Bezold's "triad" are present in most cases of chronic catarrhal deafness and do not at all indicate, as do this Lowenburg sign and the Schwartze flush of the promontory, a true osteoporosis of the labyrinth capsule.

When employing the catheter for each ear from the one nostril, I have been amazed that many experienced aurists have hailed it as a new method, although it was used by Cleland and the earliest exponents of catheterization through the nose; yet it was seemingly forgotten by Voltolini and others, whose removal of nasal spurs and other irregularities was mainly to facilitate the introduction of the catheter rather than to relieve the irritation and improve the ventilation of the obstructed naris. After using the catheter it should always be wiped with a tuft of clean cotton, so that any blood upon it may give evidence that the mucosa has been wounded and warn against any vigorous nose blowing or Politzerization which might cause emphysema. I am almost afraid to state that in 40 years I have never seen emphysema caused by myself or any of my assistants, even when the catheter was entrusted to undergraduate hands. Yet I have been told that this ball and socket catheter inflation is too rough to be safe! I hold that catheterization is a procedure, not "an operation" —there should be no real pain or bloodshed; and I strongly advise against cocainization.

The use of fluids per catheter, whether introduced by the atomizer or by a dropping tube to be sprayed up by the blast of the Politzer bag, has as one of its major values, the exciting of swallowing efforts as some of the liquid escapes into the throat; for the floor of the eustachian orifice is formed by the thinly covered lavator palati muscle, which lifts the catheter beak snugly into perfect position, while dilating the tube for the readier admission of the inflation.

In Politzerization it is usually claimed that the pressure must be equally towards each ear and that we must turn to the catheter if we wish to lateralize the effect. Aside from the safeguarding of one ear secured by closing its meatus with the finger, which furnishes a cushion of slightly compressed air to support the drumhead and even transmit a trace of counter pressure down the tube, one rotates and inclines the head to meet the finger and thereby tends to compress this side of the pharynx and tube mouth, while dilating the opposite. Furthermore, the air tends more strongly toward the opposite tube while passing by the nearer one; so it is one of the many things in otology which "go by contraries" that preponderatingly to inflate the right ear we should use the left nostril for the Politzer bag.

As a prelude to any form of inflation, the spraying clean of the tube-mouth region should be supplemented by mopping, more thoroughly to cleanse it. In this we do well to remember, as Harrison Allen taught, that the lateral pharyngitis points definitely upward to trouble of this part of the posterior nares, which in using his applicator bent at about a right angle we gently but efficiently reach, if we bear in mind the jutting out of Passavant's cushion to complete the closure of the pharynx in swallowing. Unless this is fully heeded we may fail to understand the formation of crusts which lodge above it and constitute Thornwaldt's disease; or we may injure its thin edge in forcing through it rather than past it to mop the vault. Squeezing out the excess of medicament is especially needful if we have the patient expose the throat by inspiration, lest its unintended laryngeal application be too irritating. It is hardly inappropriate to call attention, as did Harrison Allen, to the value of the aneurism needle as explorer of the tonsil crypts and as our safest instrument for

opening quinsy or postpharyngeal abscess. In quoting Harrison Allen it may be well to recall that he advised more than thirty years ago the divulsing of the bands of adhesion in Rosenmueller's fossæ, to which Myles called attention some fifteen years later, Jervey later still; and now the osteopaths have discovered—so that by their manipulation at the front of the vertebra they claim to cure tinnitus, deafness and all aural ills. I have found it occasionally quite useful during these thirty years.

Douching the ear is one of the simplest of aural measures; yet one rarely done well, if we may judge from the writings on the subject. Probably it is regarded as too easy to be worth discussing, a mere nursing matter hardly worthy of the surgeon's attention; yet not always relegated to the more conscientious care of a nurse. Many warn against its use: and as they seem to misuse it, the warning is wise. Politzer and others who speak of the temperature at all mention 88°. (Luke must have been rather coldblooded if 88° is "lukewarm.") A general bath should be 105° to feel "hot" to the body surface—to the inflamed ear 105° may hardly miss feeling cool, since the local temperature may be at least as high. Yet douching is an admirable way of cleansing, removing wax or other foreign bodies (wax has no more right to be in the depth of the canal than shoe buttons or cherry stones), but especially of applying heat, our best and most penetrating astringent, to the inflamed ear; and 115° is usually the best degree—bearable yet positive. Tediously to test the fluid with the thermometer is needless if the surgeon will throw the jet upon the thin skin between the fingers; here 110° is hot, 112° very hot, at 115° the hand tends to pull away, at 117° most persons snatch away the hand. Much of the impression of heat, however, depends upon the rapidity with which it is applied. Our tissues are poor conductors, not truly nonconductors; slow drops of boiling water can be tolerated, while a rapid flow at 115° may seem hotter. We must begin gently with any douching, although having it uncomfortably hot, therefore, avoiding any fright to the patient; then as he becomes confident and the fluid in the bottom of the canal tends to lessen the penetration of the hotter water, we can press our syringing more forcibly. Any air bubbles

make the flow intermittent and jarring—so the syringe should be emptied of all air if he is to be tolerant of force, which is the equivalent of speed of flow and vigor of impression. Quantity is almost as important as degree in the effect of our caloric, so a pint or two is usually requisite. This is tedious except with the gravity douche, and the intermittence of the bulb or piston syringe is a drawback; but with two bulbs, one filling while the other is being emptied, a virtually continuous flow can be secured. The Fowler "suction bell" has its advantages for this as for the Barany douching—which should remind us to look out for vertigo and nystagmus, which usually appear before any moisture of the brow and temple.

LXXIV.

ACUTE ABSCESS OF THE LATERAL WALL OF THE LARYNGOPHARYNX.

By Charles W. Richardson, M. D.,

Washington.

Clinically we occasionally meet with conditions which we have not seen described in the literature, which seem to us unusually rare, and which cause us either to doubt our clinical acumen, or wonder if such experience has not been the experience of others who have for various reasons not given description to their findings.

The condition which I propose to report briefly today is the occurrence of an abscess in the laryngopharynx. In this region, occasionally, we have abscesses directing their way down from the peritonsillar pus formation. In this type of laryngopharyngeal abscess we have the easily recognized relationship with the coexisting peritonsillar abscess. The form of laryngopharyngeal abscess which I shall describe is primarily an abscess of this region, with an absolutely normal faucial region. In the past ten years I have seen three primary abscesses of this region, two of which I have observed in the past eighteen months. From the clinical account of a case which terminated fatally in the practice of a colleague, I believe that he had to do with an abscess of this character.

The etiologic factors which enter into the formation of an abscess in this region have been difficult for me to elucidate. It is possible that a lymphatic gland, numerous in this region, might have become infected, or some article of food containing a foreign body might have slightly incised the mucosa and thereby produced infection.

The symptoms are quite characteristic. The onset is quite sudden. Pain is usually the earliest symptom. The pain is very intense and is located just below the angle of the jaw, over the great cornu of the hyoid bone. Tenderness to pressure in this region is very marked. Pain in swallowing is

exquisitely pronounced. The pain is so great as to render sleep fitful and unrefreshing. There is a profuse secretion of mucus, but no insalivation. There is a noticeable tumefaction to the touch on the second or third day, which no doubt would become markedly visible did not spontaneous or artificial evacuation take place. Respiration is not affected and the voice is usually normal. Chilly sensations are present from time to time. The temperature is septic, but with only a moderate wave. The patient is decidedly uncomfortable, restless, anxious and entirely disturbed about the issue of his case.

Local manifestations: The faucial regions appear normal; no tenderness about tonsils. Depression of the tongue is painful. Through the use of the laryngeal mirror a distinctive swelling can be observed protruding inward in the lower pharynx on a line with the fossa navicularis. This region has a slightly congested appearance. On the second or third day distinctive pointing takes place, from which spontaneous rupture with evacuations may take place, or through which an incision may be made which will result in the evacuation of the abscess.

In conclusion I wish to make several observations:

1. The sudden onset.
2. The severity of the symptoms; in particular, the pain.
3. The absence of tonsillar infection.
4. Location well below the lower border of tonsil.
5. The uncertainty of its etiology.
6. As rare as it seems in my clinical work, I am sure that many of the membership have seen this type of faucial infection and may help to elucidate what is still unclear in my report.

LXXV.

CHLOROMA WITH SPECIAL REFERENCE TO EAR, NOSE AND THROAT MANIFESTATIONS, WITH REPORT OF TWO CASES.

By W. W. Pearson, M. D.,

Des Moines, Iowa.

Chloroma is a rapidly growing and very destructive new growth usually occurring in the young. It arises from the mother cells of the white corpuscles and is characterized, anatomically, by the formation of tumor masses, some or all of which have a greenish color. These have a predilection for bone and are most frequently found in or on the bones of the skull, but other tissues are also invaded. Usually the tumor cells are found in large numbers in the blood. Histologically chloroma may be divided into the myeloid or lymphatic types, according to the kind of leucocytes present. Clinically the condition most frequently manifests itself by tumor formations in the orbital or temporal regions, by swelling of the face with exophthalmos, tenderness over the affected bones, impairment of nerves due to pressure, a rapidly increasing anemia and cachexia with fever and an hemorrhagic diathesis.

The first description of this disease was by Allan-Burns[1] in 1821, while King[2] in 1853 was the first to name it chloroma. Since then about 100 case reports have appeared in the literature. Many of these are accompanied by most painstaking autopsy reports and pathologic discussions.

It was my good fortune to be present at the autopsy in 1892 of the first case reported by Dock and Warthin.[3] It so impressed me that when I saw my first case the possibility of its being chloroma occurred to me. (It was not, however, until the clinical examination by Dr. Walter L. Bierring that the diagnosis was made. I was called in council because of the orbital condition and ear complications.)

A second case was referred to me and admitted to the Iowa Lutheran Hospital, March 18, 1919, with a diagnosis of orbital abscess. Having recently seen the other case and

failing to discover an origin for an orbital abscess, I immediately secured the results of successive blood examinations and became convinced that I was dealing with another case of chloroma. The subsequent history of the case, with death within a few weeks, was also in keeping with the diagnosis. Unfortunately I was defeated in my efforts to secure an autopsy.

It occurred to me that it would be of interest to this society to report the two cases which came under my observation, and to present excerpts from the recorded cases of chloroma which suffered involvement of the ear, nose and throat. (The autopsy findings in my first case with the chloromatous masses in the nasal sinuses, middle ears and right mastoid prompted this consideration.)

Case 1.—S. C., white boy, aged 12, entered the Iowa Methodist Hospital December 31, 1918.

Present complaint: The patient came to the hospital complaining of pain in the right ear and at the base of the brain. He is unable to close his right eye or to move the right side of his face. He further complains of hearing poorly on both sides, and states that he cannot see as well as he did a few weeks ago. He has pain in the right eye and in the lumbar region of his back.

His present illness began two weeks after he had recovered from an attack of influenza which went through the family in the early part of November, this year. He first noticed a "ticking" sensation in the left ear, and then a "thundering" in the right. Soon after this his right ear became painful. During the first week in December the patient observed that his right eye was black and that the eyeball was bulging. A few days later he noticed a bloody discharge from his nose. This lasted for a couple of days. During the time of the discharge his eye felt better. The pain in the right ear has been continuous, and his eyes have become more and more prominent. All of his symptoms have gradually increased.

Past history: He had infantile paralysis at the age of two. All extremities were involved, but he regained the use of all but the left foot, which remained partially paralyzed. Two and one-half years ago the right foot also became partially

paralyzed, and since that time he has never been as vigorous as other boys. But he has continued his school work. His appetite is good; he sleeps well. But since his entrance into the hospital his bladder has had to be catheterized. His father states that he was a full term baby and has been an average child in every respect. Family history negative.

Physical examination (W. L. Bierring): The boy is somewhat emaciated. The skin is pale and has a light yellow tinge. He is rather restless in bed. He cannot hear the ordinary voice, but when one speaks moderately loudly he understands questions and replies quickly and intelligently.

Head: The head is well formed. There is a moderate growth of hair. The base of the skull and the back of the neck are moderately tender to pressure. There is also tenderness over both mastoids. This tenderness is slightly more marked on the right side. Both eyeballs protrude markedly —the right more than the left. The right lids are somewhat swollen, purplish in color, and the venules of the lids on both sides are prominent. He cannot close the right eye completely. The right pupil is moderately dilated. The left pupil is normal, and both react to light and accommodation. There is marked engorgement of the lateral conjunctival vessels of the right eye. Nystagmus is absent. The eye movements are limited. He cannot read as well with the right as with the left eye.

There is moderate discharge from both ears. The nose is negative. The teeth are in fair condition. The tonsils are of moderate size. The tongue when protruded points to the right. The patient cannot wrinkle the right side of his forehead nor retract the mouth to the right. The superficial veins of the neck are distended. A few hard lymph nodes can be felt in each anterior cervical region. There is a moderate rigidity of the neck.

Thorax: The lower one-half of the sternum is somewhat depressed. The costochondral junction on the left side between the third and fourth ribs is slightly prominent. There is tenderness to moderate pressure over the upper one-half of the sternum. The lungs show nothing abnormal. There is a slight systolic murmur heard best at the base, but also

at the apex. Nothing abnormal can be detected by abdominal examination. The feet are in the position of talipes equinovarus. The reflexes of the upper extremities are normal. The patellar are absent, so are the Achilles. The Babinski sign is absent, but there is a suggestion of a positive Kernig on both sides.

Special examination: This examination, made December 31, 1918, showed marked congestion in both the middle ears. The right ear bulges. The hearing is decreased, a watch can be heard one inch away on the left side, and on contact on the right side. Paracentesis of the right ear drum was made and resulted in a small amount of a heavy discharge. Later this discharge became more fluid. The patient complained of pain over the right mastoid, which was sensitive to pressure. This continued throughout the illness. Radiograms taken on several successive days revealed a cloudiness of both mastoids, more marked on the right.

Eyes: The veins of both retinæ are moderately congested. Before the fatal termination multiple hemorrhages appeared in both retinæ. A marked exophthalmos was present in both eyes, more marked on the right side. A double optic neuritis was also present.

The patient continued to go downhill and died on the last of January. At the entrance there were 2,800,000 red cells and 24,000 whites, with hemoglobin, 45 per cent. On the 13th of January the white count reached 41,000. The day prior to death it was 18,000 with hemoglobin 20 per cent. In the differential count a rather large mononuclear cell, resembling myelocytes, predominated. This type of cell comprised between 90 and 100 per cent of all those found in the blood toward the last. While in the hospital the boy ran a continuous fever, which reached 104 two days before his death. He also developed a cystitis and had to be catheterized. An occasional granular and hyaline cast was found several times.

Autopsy report (Julius S. Weingart, M. D.): The body is that of a white male child, 57 inches in length. It is markedly emaciated. A mottled purpuric rash covers the abdomen and the lower part of the chest. A few purpuric blotches are present on the arms and on the lateral sur-

faces of the thighs. A bilateral talipes equinovarous is present, and also a dry purulent discharge from the right auditory canal.

Head: The dura in the cranial cavity is found adherent along the sagittal suture over an area 3 to 5 cm. in width and 8 to 10 cm. in length. It is also markedly thickened (in places to the extent of .5 cm.). This thickening is somewhat irregular and nodular in character. It is firm and thickest at the sinus itself, thinning out as it extends laterally. The color is greenish with scattered, dark, hemorrhagic areas. There is some engorgement of the cortical vessels in both right and left occipital regions of the brain. In the dura lining the left half of the posterior cranial fossa a mass about 2 cm. in diameter and 6 to 8 mm. in thickness, circular in shape, and slightly elevated above the surface of the dura, is seen. This has a distinctly greenish tinge. Examination of the venous sinuses shows nothing except that the wall of the right lateral sinus contains thickened areas similar in character to those seen about the superior longitudinal sinus. A small loose clot is found in the lumen. The interior of the right mastoid bone and the middle ear is filled with a gelatinous greenish mass. No definite pus is seen. Similar material also occurred in the sinuses of the sphenoid and in the right ethmoid cells. The brain itself shows nothing abnormal.

Thorax: A large flat greenish mass is seen on the posterior aspect of the sternum. This is of a greenish color, the most chloromatous of any of the tumor masses found. It is 5 to 6 cm. in width and .5 to 1 cm. in thickness, and extends from the manubrium sternum to the xiphoid cartilage. A similar flat greenish growth is found on the right vault of the diaphragm. The pleuræ and pericardium show a number of petechial hemorrhages. In the upper part of the lower lobe of the left lung there is an irregular area of partial consolidation. The heart and great muscles are normal. The mediastinal lymph nodes are not enlarged.

Abdomen: The gastrointestinal canal is negative. No definite enlargement of either mesenteric or retroperitoneal lymph nodes is present. The spleen is not enlarged. It weighs 7.5 ounces. The surface made by section has a reddish, waxy appearance. The liver is normal in size and weighs 3 pounds

3 ounces. The surface made by section appears normal, with the exception of some white striæ running along the blood vessels. Both kidneys are markedly enlarged, weighing 7 to 7.5 ounces and measuring 13 cm. by 6.5 cm. They are both white in color and mottled with hemorrhagic areas. The surface made by section shows a greenish cortex with numerous small hemorrhages in the pyramids. The bladder contains one ounce of purulent fluid. Its walls are extremely congested. Examination of the pancreas and adrenals is negative. The bone marrow from the center of the shaft of the femur is red and gelatinous. Microscopical examination of the wall of the longitudinal sinus shows the thickening to be due to a tumor mass composed of cells, round, or somewhat polygonal in shape, and arranged for the most part in strands between the fibers of the small amount of connective tissue which is present. These cells have nuclei, some of which are pyknotic, others have a loose chromatin network; the nuclei fill most of the cells, so that the amount of cytoplasm is very small. This latter is in many cases acidophilic. The cells are largely mononuclear, but here and there a cell with two nuclei or with a lobed one is seen. The size of the cells varies a good deal. No mitotic figures are found. Sections from the other tumors show precisely the same histologic picture: that of a cellular mass composed of strands and rows of mononuclear cells. The liver shows foci of similar character scattered throughout. Here in addition, a few very large multinuclear cells are seen which resemble closely the megalokaryocytes of the marrow. The normal histologic aspect of the spleen is almost lost. The splenic bodies are very small. Often only a small group of lymphocytes can be seen here and there around the central artery. The rest of the spleen is packed with cells similar in type and arrangement to those seen in the tumor masses. Here very few red blood cells are seen. The marrow is replaced by the tumor cells. The kidney shows large areas where the tumor tissue has obliterated all the normal markings. In places the convoluted tubules and glomeruli are intact, but even here the tubular epithelium shows evidence of degeneration. Numerous areas of hemorrhages occur throughout the kidney substance.

Diagnosis: Myeloid chloroma.

Case 2.—R. B., male, aged 9, entered the Iowa Lutheran Hospital, March 18, 1919, with a possible diagnosis of orbital abscess. On account of my absence from the city, the patient was seen first by my assistant, Dr. Doane, who repeatedly examined the nose but failed to find any evidence of an infection. On March 24, Dr. J. T. Priestley was called in council. According to his report, the patient had been ill for several weeks, complaining of headache, general weakness, and aching of his limbs. More recently his vision had become somewhat disturbed. On examination the patient appears bright and takes a lively interest in things about him. Examination of the chest and abdomen reveal nothing abnormal. The long bones are all sensitive to pressure. The patient is unable to walk, but enjoys going about in a wheel chair. During his stay in the hospital the temperature varied between 99 and 102, pulse between 90 and 120, and respiration between 20 and 30. The urine was negative. The blood examination showed 2,000,000 red cells at entrance; before he left they dropped in number to 1,500,000. The whites were 33,000 at entrance, at one time reaching 56,000; 98 per cent of them were large mononuclear. Hemoglobin, 35 per cent; Wassermann negative. Radiogram of temporal bone showed nothing abnormal.

I examined the patient on March 29 and noted the following: The hearing is reduced on both sides. Examination of middle ear reveals nothing. There is a marked proptosis of the right eye, while the left is decidedly more prominent than normal. Both retinæ show marked neuroretinitis. Pupils react to light and accommodation; the eye movements are but little restricted. Examination of the nose is negative. Before leaving the hospital, hemorrhage of the right retina occurred, and during his last days in the hospital several nasal hemorrhages took place; none were dangerous.

The boy remained in the hospital eighteen days, when he was taken home. He died about two weeks after.

Review of Literature.—Alexander[4] states that he found lymphatic tumors in the labyrinth in leukemia, lymphosarcoma and chloroma.

Benjamin and Sluka[5] mention the frequency of deafness and seventh nerve paralysis in chloromatous subjects. The

symptoms are due to the tumor deposits in the temporal bone.

Graupner[6] relates the case history of a boy with a right-sided facial paralysis and vertigo. Both external canals were dropped and a slight discharge of fetid pus exuded from the right ear. The mastoid on this side was painful. Perception of the lower tones was lost. The patient ran a slight fever. The right mastoid was operated upon, and a green mass was found within. The facial canal was involved for a distance of one centimeter. The patient died nine days later, and among other chloromatous changes found was a tumor formation in the inner auditory canal exerting pressure on the seventh and eighth nerves.

Lehndorff[7] prefaces the recital of two case histories of chloroma, stating that in many cases the usual clinical and blood picture is not present from the first. The two cases reported by him entered the hospital as septic diphtheria. The pharyngeal appearance, glandular enlargement, pulse, and temperature were those of diphtheria. One was a boy of 4½ years of age, who had been ill about fourteen days, suffering from a high fever, malaise and swelling of the submaxillary glands. Later he had a very foul breath, difficulty in swallowing, and frequent hemorrhages from the gums and skin and an increasing anemia. Examination revealed the left tonsil greatly swollen with a deep area about the size of that of a dollar covered with a foul gray green exudate that extended to the pharyngeal wall. At no time were diphtheritic bacilli found. There was a remission in the process and the temperature dropped to normal. The pharyngeal condition improved and apparently began to heal. A week later a short turn for the worse was noted, with high fever, increased anemia, and the tonsillar ulcerative process started anew, with increased glandular swelling in the neck. The child was removed from the hospital, but returned a few days later because of a developing dangerous pharyngeal stenosis. At this time the tonsils, palate and pharynx were covered with foul yellowish membrane. The enormous swelling interfered with speech, swallowing and breathing. After a few weeks' illness death ensued. An autopsy by Störk showed that the symptoms noted were due to chloroma.

Lehndorff's second case was that of a nine year old boy who began to suffer from malaise and fever in June. On June 12 he complained of sore throat. Diphtheritic antitoxin was injected on the 20th, and he entered the hospital with the diagnosis of septic diphtheria. The patient was emaciated and pale, with many old and recent subcutaneous hemorrhages. A high fever and rapid, poor pulse were present, and there was a systolic blowing heard all over the atria of the heart. The liver was soft and extended three fingers' breadth below the ribs. The spleen was relatively small, hard and palpable. The appearance of the throat suggested septic diphtheria. The breath was very foul; the tonsils were reddened and swollen; the uvula was swollen. On the left side there was a dirty, grayish yellow ulceration which extended over the left pillars. The exudate on the posterior pillars was thick, yellow and uneven. About the exudate there were hemorrhagic areas. These also occurred in the pharyngeal mucous membrane and on the cheeks. The glands under the jaw were small and painful, and the covering skin was edematous. Smears from the exudate showed diphtherialike bacilli, cocci in chains and clumps, fusiform bacilli and spirilla. The culture showed only staphylococci and streptococci. During the following days new hemorrhages appeared in the skin and mucous membrane, and the pharyngeal condition grew worse. The glands under the sternocleidomastoid were enlarged. The spleen and liver increased in size and grew softer. The following week the throat and pharyngeal condition remained stationary, but the temperature increased. The eye backgrounds were normal, and there was no exophthalmos. The bones were not painful. There was an occasional nasal bleeding. The axillary and inguinal glands were slightly enlarged during the last days of his life. The disease ran its course in about four weeks. The autopsy, performed by Weisner, showed beside other changes a chloromatous color in the glands of the neck and thorax. The histologic findings in this case were a surprise and indicated their importance in making the diagnosis.

Walls and Goldsmith[8] reported a case of a five year old boy, whose illness covered a period of three months. The pathologic report showed chloromatous deposits in the fol-

lowing locations: orbits, frontal bones, nasal sinuses, temporal bone, external surfaces of the parietal bones, attached to the cerebral and spinal dura mater, kidney, liver and posterior surfaces of the sternum. One large chloromatous tumor was found between the eyes. This seemed to push apart the two halves of the face. The distance between the inner canthi was 3 cm. The pathologic findings in the larynx are of especial interest. "The upper part of the larynx shows necrosis as does also the epiglottis and fauces. The nasopharynx exhibits necrosis of the mucosa and submucosa and of some of the layers of striped muscle. Between the bundles of muscle fibers and even between the individual fibers are seen mononuclear cells, some of which are ordinary lymphocytes, but many of them are large cells supplied with large pale nuclei."

Hirschfeld[9] reported the case of an eight year old girl who suffered from a growth beginning in the right antrum, of about six months' duration. When seen by him it exerted pressure outward and downward. The right alveolar process was lower than the left. The nose was crowded to the left. The seventh nerve was intact. The covering skin was cyanotic. The entire tumor was sensitive to pressure. An extension of the tumor up into the orbit and back of the eye produced a marked proptosis. The upper lid covered the eye but the lower one was ectropic. The eye was blind (the result of optic atrophy). The blood examination decided the diagnosis clinically as chloroma. Hirschfeld emphasizes the importance that the blood examination has in the presence of malignant tumors.

Merril and Bedell's[10] case occurred in a boy of eight years with the following ear complications: The external canal of the right ear was moist and almost occluded with inspissated pus. The posterior superior wall was found bulging and moderately congested. The tympanic membrane showed a calcified area in the lower posterior quadrant. Above this was a small perforation through which a yellowish exudate escaped. The left ear showed a similar condition without the bulging posterior wall. Both mastoids were distinctly tender to pressure. No autopsy was permitted in this case, but a chloromatous tumor was uncovered in the left orbit under anesthesia a few days before death.

Bruce[11] reports the case of a married woman, aged 38 years, who suffered from violent neuralgic pains over the temporal region, cheek and upper jaw of the left side. She first noticed a swelling of the gum of the upper jaw in the region of the first bicuspid tooth of the left side. A day or two later she noticed a similar swelling at a corresponding point on the opposite side, and simultaneously a swelling made its appearance around the first bicuspid of the lower jaw on the left side. The pain subsided, but the swelling continued to increase. On examination, about six weeks from the onset, a marked swelling of the gums was seen. There were swellings under the jaw on both sides. The glands were fairly movable and not tender to touch. The other glands of the neck were not involved. The skin was swarthy. The breath was very foul. The teeth were all in line and not loose. The swelling of the gums was very marked, spreading between the teeth, and were convoluted, dark purplish red in color and covered with epithelium. They were firm to touch and did not bleed. The tonsils were not enlarged. Four days later there was an ulceration and sloughing over the first right molar above. A piece was removed for examination, and the pathologist reported that it was probably lymphosarcoma. About four weeks later the patient began to run a septic temperature from 100 to 104 degrees, with a pulse of 110 to 140. The swelling on the left side of the neck rapidly increased in size, as did also the growth on the gums. The breath was very offensive. There was much salivation and she was getting progressively worse. Pus was exuding from around the teeth and gums. It was thought that absorption of this pus was responsible for the temperature. An operation was undertaken with the object of not only removing the growth but of getting rid of the septic condition around the growth and teeth. She survived the operation only a few hours. The autopsy diagnosis was chloroma.

Pfeiffer's case[12] was that of a boy four years of age, who previously had fallen and cut the left side of his forehead, which healed promptly. Several weeks later he suffered with an inflammation of the other ear with a purulent discharge. Following this, the child continued ill with loss of appetite, and became pale. About two weeks later proptosis of the left

eye developed. A double papillitis with unequal pupils and limited motion of the eyes were noted. Examination of the left ear was negative, while a small perforation in the right upper posterior quadrant was present. Hearing on the right side was markedly reduced. The temporal and malar bones of both sides, and especially the left, were prominent. Proptosis of the right eye developed. A questionable diagnosis of tumor of the ethmoid or sphenoid and thrombosis of the cavernous sinus following the right middle ear infection was made and the skull opened external to the outer angle of the orbit and above the malar bone. No tumor or occlusion of the veins was found, although exploration with the finger reached the middle line and the region of the pyramid. The wound showed no tendency to heal. There developed a cerebral hernia, the bone margin became necrotic and death resulted after a four months' illness. At autopsy many yellowish green tumors, .5 cm. thick and about the size of a half dollar, were seen on the outer side of the dura. They were especially numerous along the wall of the longitudinal and transverse sinus. They were not attached to the bone. The entire right parietal bone and mastoid were found infiltrated with a yellowish green mass. The left tonsil, although not enlarged, showed the light greenish color. The submaxillary glands of both sides and collections about the larynx showed the same greenish deposits.

Saganuma[13] reported a case in which both maxillary antri contained tumors.

Wynter and Laken[14] report a case with the following autopsy findings: "The face is distorted. There is a considerable bulging of the cheeks due to immovable swellings in the superior maxillæ. Proptosis is present and the bridge of the nose is broadened and flattened. The skin below the eyes and upper portion of the face shows a greenish purple discoloration. Bleeding has evidently taken place from the gums and there are thickenings along the inner side of the lower jaw."

Pfeiffer discusses the clinical diagnosis of chloroma under four headings: First, there is painful exophthalmos with secondary optic atrophy. This is one of the earliest symptoms. Heyden found this to be the earliest symptom in 31 out

of 42 cases. In about one-fourth of the cases of chloroma the growth has its origin outside of the skull. Hence, exophthalmos is absent only in those cases in which the bones of the skull are not involved in the process.

The second series of symptoms are due to the growth of the chloroma in the temporal bone, causing middle ear infections, hemorrhages, tinnitus, reduced hearing and swelling and painful mastoids. These symptoms were present 25 times in the series of 42 cases. They are bilateral. According to Körner, chloroma is relatively more often met with in the external canal than any other tumor involving the ear. Disturbance of equilibrium resulting from chloromatous involvement of the static portion of the internal ear has been noted by Körner, Weinberger and others. Impaired function of the seventh nerve due to chloromatous infiltration has been repeatedly observed. Along with the ear disturbances there frequently appear chloromatous deposits under the temporal muscles, on one or both sides. Similar growths frequently occur in the nose, giving rise to hemorrhages which are sometimes fatal. In cases where the nose has been obstructed autopsies have revealed chloromatous growths in the sphenoid, ethmoid, antrum or pharyngeal vault (according to Körner, Chiari, Behring and Rosenblath). Strange to say, intracranial pressure symptoms, slowing of the pulse, vomiting, etc., have never been recorded, although growths along the venous sinus in the dura have frequently been present.

Thirdly, there is marked anemia, especially in the latter half of the disease. Many times the picture is that of a typical acute leukemia with an enormous increase in the number of white blood cells. The leukemia may appear suddenly shortly before death. (Rosenblath and Weinberger.) Coincident with this appear many large and small glands along the neck, mediastinum and abdomen. They are made up of one or more green nodules. Many times the spleen becomes swollen so that a diagnosis of leukemia is made.

The fourth point in diagnosis is the age at which the disease appears. Among 33 cases of chloroma, 28 appeared between the ages of one and twenty-five. Five cases were between the first and fourth years, eight between six an ten years. In cases of advanced age the growths are seldom found on the

skull. The number of male sufferers is greatly in excess of the females.

The disease usually runs a short course. Death ensues in the great majority of cases within three to four months. The direct cause of death is hemorrhage or general marasmus.

In conclusion, I believe it is important for the specialists in head work to be familiar with this condition in order that unnecessary operations may be avoided.

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LXXVI.

CONCERNING THE QUESTION OF JUGULAR LIGATION† IN SINUS THROMBOSIS.*

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Thirty years ago Horsley[1] of London advocated the ligation† of the jugular vein in cases of infective sinus thrombosis, but gave as the indication for the procedure the presence of metastases, hoping thereby to prevent further infection.

Ten years later, when reporting two cases of infective thrombosis, C. Barck[2] of St. Louis wrote that the question upon which no unanimity of opinion has been reached, is the ligation of the jugular vein, and forthwith stated his belief that the primary ligature of the jugular vein is justified in all cases of sinus thrombosis.

Between these two extreme views, the ultraconservative view of Horsley, who waited for the appearance of metastases, and the ultraradical view of Barck, there are all sorts of opinions recorded. In 1903 Politzer[3] (p. 614) wrote that the question of ligating the internal jugular vein is not yet settled, and as late as a quarter of a century after Horsley's original recommendation, Kerrison[4] is forced to the conclusion that concerning the question—in what class of cases is the sinus operation alone—i. e., without jugular ligation or resection indicated? otologic literature throws little light.

What then are some of these divergent views? By whom held? And how justified? After critically reviewing, in the light of present knowledge, the principles upon which these various views are based, the writer will append his conclusions which seem to be justified from personal experience

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*Read before the Pacific Coast Oto-Ophthalmological Society, July 29, 1920.

†The term ligation as used in this paper refers to any jugular operative interference, whether simple ligation or ligation with resection.

and a study of the literature. The various views to be discussed are:

1. Those in favor of ligation in all cases.
2. Those opposed to ligation in all cases.
3. Those in favor of ligation only when free bleeding does not occur from the jugular end of the sinus.
4. Those in favor of ligation only when rigors and fever continue after the removal of the thrombus.
5. Those in favor of ligation only after the development of metastases.

I.—THOSE IN FAVOR OF LIGATION IN ALL CASES.

Barck[2] took this stand, claiming it was almost impossible to ascertain during the operation the true state of affairs in the bulb and sinus.

Kerrison[4] is of the same opinion and goes one step further in stating that the sinus operation itself may produce a traumatic thrombus even when no thrombus previously existed. He states his views as follows:

"Theoretically this question is easily answered by the statement that it (the sinus operation alone) is indicated in cases in which the physical evidences on opening the sinus are of a circumscribed lesion situated well above the lower end of the vertical limb. But how are we to determine such limitation? Suppose, for example, that we operate upon a case in which symptoms of periodic septic absorption have been present, and that on opening the sinus we have a free bleeding from the direction of the bulb, and a demonstrable clot toward the torcular end. Snch a case would seem to present ideal conditions for the sinus operation alone. But can we be sure that a secondary infection at the bulb does not already exist, or that in the manipulation incident to the opening of the sinus, its compression below and the removal of a septic clot above, we have not sown the seed for a septic thrombosis below? Or suppose that we find absolutely no physical evidences of a clot; can we from this conclude that a nonobstructive parietal clot does not exist in the bulb? Personally I feel that when we have opened the sinus to the extent necessary to explore its lumen for the presence of a suspected clot, we have incurred the risk of producing a traumatic thrombus, even though none has previously existed. For this reason and

those stated above, I believe that when we have assumed the responsibility of opening a sinus on account of symptoms of septic absorption, we should in every case further safeguard the patient by ligation or resection of the jugular vein. As to which operation should precede the other—i. e., the exploration of the sinus or the ligation or resection of the vein —ordinarily I do not believe that it greatly matters. When, however, the patient's condition is so profoundly septic and his vitality so greatly reduced that the addition of any fresh septic material to that already circulating in the blood might turn the scale unfavorably, I should say that ligation of the vein should precede the opening of the sinus. In any case, and whichever order of precedence is observed, the sinus should always be freely exposed and ready for exploration before the vein is operated upon."

To the above I would apply the author's own remarks concerning anther question (the forcing of septic material toward the torcular end from manipulation below): "These contentions while theoretically correct are not in my opinion of great practical significance." And this is stated because of the numerous instances of cure in which the sinus operation has been performed without disturbing the jugular vein. As Prof. Da Costa has once remarked, the law of probabilities must always be taken into consideration.

Politzer[5] quoted Bergmann, Forselles, Lane and Zaufal as advocating ligation of the vein before opening the sinus, but only, however, in cases in which a septic thrombosis is found after the sinus is exposed, the reason being that by ligating the vein, septic material and particles of thrombus which have been loosened by the operation are supposed to be prevented from entering the blood stream. Of course this is only partially true, because while the main channel is thus occluded, there are left collateral venous channels through which infection can be carried.

In the discussion of Dr. Whiting's paper,[6] Sheppard states that his experience leads him to approve of previous ligation of the jugular, and because more recent statistics show that there is the largest percentage of recoveries where the vein is tied before any other operative procedures are resorted to.

Lilienthal in the same discussion said the ligation of the

vessel should precede any chiseling of bone or any palpation of the sinus or the soft parts about it, so that in the event of the dislodging of septic material from the thrombus during the necessary operative manipulations, the door of entrance to the circulation might be closed in advance.

Jones[15] sent a questionnaire to 100 of the leading otologists of the country, and of the 19 who responded 13 favor jugular ligation in every case of lateral sinus thrombosis.

Without attempting to make an exhaustive review of the literature, I believe sufficient authorities have been quoted to make it incumbent on anyone opposing the ligation in all cases to fortify himself well with facts if he hopes to have his views justified.

These facts I now shall attempt to present:

II.—THOSE OPPOSED TO LIGATION IN ALL CASES.

When such authorities as Politzer, Körner and Bezold oppose the ligation in all cases, we must at least admit that there are two sides to this as to every other question.

Politzer[3] writes as follows:

"The favorable results after ligation of the internal jugular vein quoted in literature cannot be taken as being due to this operation, as recovery often occurs after cleaning out the sinus without ligation of the vein. That, in spite of ligation, septic matter may enter the blood current from the infective focus in the sinus through collateral venous channels is beyond all doubt. A condition which also contraindicates ligation of the jugular vein before opening the sinus is incomplete thrombosis; in such cases ligation of the internal jugular vein causes the current of blood to flow backwards towards the venous blood vessels of the skull, by which the superior and inferior petrosal sinuses, as well as the larger venous vessels of the cranial cavity may become infected." (Jansen, Brieger.)

Allport[5] does not believe that the favorable reports of jugular ligation in sinus thrombosis need necessarily be attributed to the ligation, quoting Körner's excellent results without ligation, to which reference will again be made. Allport writes:

"I am convinced that excision of the jugular is not an operation which should be undertaken hastily, as grave circu-

latory and other disturbances are liable to ensue, and other methods of relief should first be tried, unless the patient's condition is very urgent. The literature furnishes many instances where sudden optic neuritis, edema of the brain, with hemorrhages and softening, thrombosis of other cerebral sinuses, etc., have followed jugular ligation, and it should not be forgotten that a small and insufficient jugular on the other side, totally unable to assume double duty, may be present and precipitate most unfortunate results. As a deduction from these observations, Eagleton urges that in cases of sinus thrombosis with optic neuritis, it should be concluded that the return venous flow is already seriously obstructed and that great caution should be observed in adding to such conditions by ligation."

That ligation of the internal jugular vein is not the simple procedure many would have us believe is also evidenced by the report of Stolz,[14] who adds a fourth case to the three he has found on record in which ligation of the internal jugular vein had fatal consequences. Linser found evidences of insufficiency of this vein in three per cent of 1,000 skulls examined, and this was five times more frequent on the left side than on the right.

Returning now to Körner's[10] work on 314 cases, this author opened the sinus without ligating the jugular vein in 132 cases, of which 77, or 58.3 per cent, were cured. The jugular vein was ligated before opening the sinus in 94 cases, 56 of them, or 59.6 per cent, were cured. The jugular vein was ligated after opening the sinus in 69 cases, 38 of them, or 55.9 per cent, were cured. From this analysis, Körner draws the following conclusion: "That it is best to adapt the method and extent of the operation to the finding in each case. To ligate the jugular vein in all early and late operations for thrombophlebitis of the sinus as a matter of principle seems under all circumstances not justified according to the number of recoveries without ligation."

Bezold[11] is convinced that the favorable results do not depend so much upon a certain method of operation as upon an early interference, and he quotes in support of his contention Toubert's statistical compilation of 200 operated cases of sinus thrombosis.

Toubert[12] operated upon 120 of the 200 cases within the first week after the appearance of endocranial symptoms, with 75 per cent recoveries and 25 per cent deaths. The other 80 cases could be operated upon only after the first week, and of these only 37.5 per cent recovered, while 62.5 per cent died.

After analyzing 50 case reports, Jones[15] has discovered that the mortality following sinus operation is as great where there is routine ligation of the jugular as it is where the ligation is reserved for the severe cases. For instance, in the 35 cases where the vein was either ligated or resected five died, 14 2/7 percent, while in the 15 cases where the jugular vein was not treated surgically two died, or 13 1/3 per cent. This, as you see, coincides very well with the statistics of Körner.[10]

Crockett[16] reported 60 cases from different operators in which the vein was ligated before or immediately after opening the sinus. Nine cases died, making the death rate 15 per cent.

If any further justification is need for opposing ligation in all cases, that is really supplied by Day's[13] report of a spontaneous cure of six cases of unrecognized sinus thrombosis which were accidentally discovered during operation, out of a total of 45 cases of thrombosis. In other words, 13½ per cent of this author's cases healed spontaneously.

Believing now that there is a justification for anyone opposing jugular ligation in all cases of sinus thrombosis, there still remains the necessity of stating, in what elective cases jugular ligation is indicated. But first we shall continue our original intention of discussing the other views previously mentioned, and in passing it may be mentioned that the authors to be quoted in this next group stand side by side with those mentioned as opposing ligation in all cases.

III.—THOSE IN FAVOR OF LIGATION ONLY WHEN FREE BLEEDING DOES NOT OCCUR FROM THE JUGULAR END OF THE SINUS.

Of the writers advocating this procedure, may be mentioned Allport,[5] Whiting,[6] Macewan (quoted by Politzer[3]), Loeb,[7] Ballenger,[8] Phillips[9] and Bezold.[11] Phillips, however, makes some exceptions and ligates (1) if clot is undergoing disintegration, (2) should patient's previous symptoms indicate pro-

found sepsis, (3) or if there is marked tenderness and infiltration along the course of the vein with enlargement of the cervical glands.

In the absence of any of the graver indications for jugular ligation, such as metastases and physical evidences of thrombosis in the jugular vein, I think this view favoring the ligation only when free bleeding fails to occur from the jugular end of the sinus, offers the most rational indication for operative interference on the jugular vein in the presence of sinus thrombosis that we possess.

IV.—THOSE IN FAVOR OF LIGATION WHEN RIGORS AND FEVER CONTINUE AFTER THE REMOVAL OF THE THROMBUS.

Politzer,[3] and Jansen quoted by Politzer, advocate this procedure, and its rationale appeals to me in those cases that have escaped the ligation at the primary operation because free bleeding was obtained from the jugular end of the sinus. This indication will take care of those few cases on which the advocates of ligation in every case base their views, namely: Those cases which are infected in the bulb and vein in spite of the fact that free bleeding occurred following the curettement of the jugular end of the sinus. But why judge all by the few? We grant that free bleeding from the jugular end of the sinus does not necessarily prove that there is no infection in the bulb and vein, but we do not grant the point that a possibility is a probability. We contend, and statistics prove our contention, that the majority of cases from which free bleeding has occurred from the jugular end of the sinus, are safe cases for the jugular vein to be left undisturbed.

V.—THOSE IN FAVOR OF LIGATION ONLY AFTER THE DEVELOPMENT OF METASTASES.

Horsley (quoted by Politzer,[3] Allport[5] and Barck[2]) was the exponent of this procedure, but I doubt very much if anyone can now be found rash enough to advocate delay to such a degree. It would be safer by far to ligate in all cases than to ligate only at this terminal stage, though, of course, an occasional cure will result, but at the price of many more than an occasional death.

From a consideration of this question from the various viewpoints thus hurriedly presented, I think we are justified

in accepting Körner's dictum "that it is best to adapt the method and extent of the operation to the finding in each case."

Personally I can see no more justification in the practice of ligating all cases than in the practice of operating upon all acute mastoid cases or of enucleating all tonsils. Let each case be a law unto itself, and until some indication other than sinus thrombosis exists let us not increase shock and lengthen the operative procedure unjustifiably.

In conclusion, may I be permitted to present the indications for jugular ligation in sinus thrombosis which I feel to be justified, from the short study that I have thus far made of this problem:

1. In the presence of metastases or when the vein presents physical evidences of thrombosis.

2. When free bleeding fails to occur from the jugular end, even though there are no metastases or physical evidences of thrombosis in the vein.

3. Finally, even though free bleeding has occurred from the jugular end, if rigors and fever continue or reappear after the sinus operation.

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LXXVII.

LATERAL SINUS THROMBOSIS, WITH REPORT OF SEVEN CASES.*

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One of the most disturbing complications of mastoiditis is furnished by lateral sinus thrombosis. This complication, always a possibility, generally gives sufficient worrk to the attending otologist so that he may not find life too motononous, at least just at that time. With this condition threatened there is usually considerable food for thought. Even with the diagnosis established and the seemingly proper course of procedure followed, there is still generally sufficient cause for concern, until undoubted convalescence is reached. Although Zaufal, in 1880, first suggested opening the sinus in a case of thrombosis, until Ballance,[1] in 1890, advocated the ligation of the internal jugular vein and shutting off the infection from the general circulation, this condition was indeed considered of the most serious nature, if not of necessarily fatal consequence. Much has been written on this disease, a great deal of which has been of inestimable value, but it may not be amiss to report the following series of cases occurring in the Oto-Laryngological Service at U. S. A. General Hospital No. 14, Fort Oglethorpe, Ga., during the year 1917-18.

Lateral sinus thrombosis may generally be considered as a complication of mastoiditis, either acute or chronic, although a number of cases of primary infection of the jugular bulb and sinus have been reported by McKernan[2 3] and others. These accompanied an acute otitis media, the pathway being directly from the middle ear to the bulb, either by dehiscence in the bony wall or by means of small communicating veins and lymphatics, or by direct absorption of bone. Other cases have been reported where there was no evident middle ear

*Candidate's thesis, American Laryngological, Rhinological and Otological Society.

disease and the sinus was found filled with pus at operation. Sauer reported such a case in 1910. However, it is quite possible that there had been some otitis media at some time previously, if unrecognized by the patient at the time.

The ratio of sinus thrombosis to mastoiditis is hard to determine. Figures vary a great deal, but there can hardly be any doubt that this complication is fairly frequent. Downey[4] reports five cases out of a series of 79 acute mastoids. Gerber[5] reports 25 in a series of 524. Welty[6] reports a series of 100 acute mastoids with only 3 cases of sinus thrombosis. Out of a series of 497 cases of mastoiditis at the Massachusetts Charitable Eye and Ear Infirmary during 1917 there were 19 cases of lateral sinus thrombosis. The seven cases here reported occurred in a series of 168 cases of acute mastoiditis at General Hospital No. 14.

Braun[7] in his work on the pathology of sinus thrombosis says that thrombosis of the lateral sinus resulting from middle ear and mastoid disease may occur in the following ways: "1. The inner table of the mastoid over the sinus may be diseased and an abscess formed between the sinus and the inner table. This results in an inflammation of the true sinus wall which eventually leads to the formation of a thrombus within the sinus. 2. The inner table over the sinus may be diseased and cause an extension of the inflammatory process to the sinus wall without the intermediation of a perisinus abscess. The phlebitis results in a thrombus formation. 3. A thrombus may form in one of the smaller veins of the mastoid and extend into the lateral sinus."

The usual course of events is necrosis of the sinus wall and destructive changes in the endothelial lining . Bacteria in the vessel wall set free toxins which pass into the blood stream. There is also an increase in the agglutinability of the blood platelets and an increased coagulability of the plasma which, with the destruction of the intima, make up the main factors in sinus thrombosis. Braun found that the bacteria were present mostly in the peripheral portions of the thrombus and that the younger thrombi contained few, if any, bacteria. This seems to indicate that the thrombus was formed before the bacteria themselves entered the blood stream. Thrombi may be either obliterating or parietal. Probably all start as parie-

tal, becoming obliterating after a variable time. It would seem that the thrombus which remains parietal for considerable time is far more dangerous to the patient than the obliterating type. A parietal thrombus is continually washed by the flowing blood stream, and, if of sufficient duration to have accumulated considerable bacteria, must needs be more of a menace, because of the septic material being swept into the general circulation, than in the case of the obliterating thrombus.

The underlying causes of sinus thrombosis may generally be considered under four heads:

1. The virulence of the infection.
2. The resistance of the individual.
3. Delayed mastoid operation.
4. Incomplete mastoid operation.

The distinction between the first two is often hard to determine, for they overlap each other with great frequency. Neither requires much comment. As an example of the first, we have the fulminating mastoiditis, usually streptococcic in its bacteriology, in which we may have operated as early as the third day after the onset of the otitis, and yet the picture disclosed at operation made our work seem tardy. Then we have the type of case where the mastoiditis is a complication of another disease which has sapped the patient's vitality. His resistance has been immeasurably lowered before the onset of mastoiditis. In the series upon which this article is based mastoiditis was a frequent complication following measles or pneumonia, and consequently low resistance on the part of the patient was frequently encountered.

Delayed mastoid operation is a frequent cause of sinus complication. Often this error is excusable. It may be impossible to make a diagnosis because of masked signs and symptoms, or a coincident disease. Furunculosis of the canal may prove a misleading factor and render the diagnosis difficult. An abnormally thick cortex may retard the development of inflammatory signs on the surface of the mastoid, and unless there be sufficient canal and middle ear signs present, cause delay in making a diagnosis of suppurative mastoiditis. As the result of coincident disease, the patient's

condition may be such that it is advisable to delay operation to the limit, even running the chance of complications.

Too frequently is incomplete mastoid operation the cause of sinus thrombosis and this does appear inexcusable. Far too often is a secondary operation required in order to insure healing after a simple mastoidectomy, and far too often does this period of healing drag out to unnecessary length because of incomplete exenteration of the mastoid at first operation. Thorough surgery would eliminate a certain number of cases of sinus thrombosis. The pathologic process in acute mastoiditis, as shown by Beck,[8] is by one of two ways: cell route, or confluent; vascular, or osteophlebitic. Especially in the case of the former does incomplete removal of the cellular or diploetic areas tend to a continuation of the process until we get necrosis of the sinus wall and consequent blood stream infection. Disregard of the cellular area just internal to the prominence of the digastric fossa and in the angle between the floor of the middle fossa and the knee of the sinus is altogether too common. These two areas are favorite sites in which to find a perisinus abscess. Cells posterior to the sinus are often overlooked and may prove potential factors in subsequent sinus complication. Too frequently a mastoidectomy is like sweeping a room but neglecting the corners. Earlier and more complete mastoid surgery would avoid many sinus thromboses.

The clinical picture of sinus thrombosis is practically that of general sepsis with frequently certain localizing signs. The picture may vary a great deal, making diagnosis increasingly difficult. Signs that are almost diagnostic when present, prove nothing by their absence. In the typical case we have a septic temperature rising to 10 or above, followed by a remission to normal or below, with daily repetitions. McKernon[9] considers temperature the most important symptom. Chills are frequently seen, following which there is usually profuse sweating and rise of temperature. McKernon gives the proportion of cases showing chills as 50 per cent. When present this symptom is very diagnostic, provided malaria has been ruled out. The pulse and respiration are usually proportionate in their increase to the temperature. With a disproportionately lowered pulse rate we must consider the possibility

of intracranial complications. We may have nausea and vomiting. The neck may be tender and palpation over the internal jugular may reveal a cordlike thickening. We may find edema in the region of the mastoid foramen and the emissary vein. Greisenger's sign, based upon the unequal filling of both jugular veins upon compression is rarely demonstrable. A high leucocytosis is to be expected, with the differential count showing a relative increase in the number of polymorphonuclear leucocytes. Frequently we may find a blurring of the fundi, although not generally seen in the earliest ages. A positive blood culture is almost diagnostic, but when negative means nothing. According to Libman,[10] all cases of sinus thrombosis show a bacteremia at some time in their course. Consequently the value of the blood culture often depends upon the psychologic moment of its being taken. The culture may be taken before the bacteremia has been established, or after it has spontaneously disappeared, or the bactericidal power of the blood may be increased so as to eliminate the organisms.

All these signs make diagnosis easy, provided sufficient of them be present. An added help is the history of a perisinus abscess or a necrotic plate found at the preceding mastoid operation. But how often it is that the picture lacks most of these signs, requiring us to base our diagnosis on only one or two of them, plus a general conception of the case and surgical judgment.

The possible end results of sinus thrombosis according to Braun[7] are: "1. The patient dies of general sepsis before the clot breaks down. 2. The clot breaks down in the center and the abscess finally breaks through the true wall of the sinus so that the abscess within the sinus communicates with the perisinus abscess. 3. The pus within the sinus breaks through the visceral wall of the sinus and produces a cerebellar abscess or meningitis. 4. The thrombus organizes and results in obliteration of the sinus. 5. Part or all of the thrombus becomes absorbed, resulting in the recanalization of the sinus." The thrombus may extend from the sinus into some of the pial and cerebral veins and result in the occurrence of spots of hemorrhagic softening of the brain. Metastatic foci in the lungs, from the breaking off of fragments of the thrombus

which are carried into the pulmonary capillaries, and foci in the joints, muscles and subcutaneous tissues, resulting from the bacteremia, may develop."

Many of these cases take care of themselves spontaneously without any operative interference, but almost without exception they run a stormy course. Undoubtedly there are many cases of sinus thrombosis which are never diagnosed as such. Day[11] reports six such cases accidentally discovered during subsequent mastoid operation. However, it is putting too much responsibility upon nature to allow this course to be pursued if possible to do otherwise. Interference as early as possible with the establishing of the diagnosis offers the best hope. The difficulty usually arises from the inability to make the diagnosis early enough. As this rests upon the clinical picture of general sepsis of a variable degree, there may be many difficulties presented. We may have a double mastoiditis and be unable to convict either side as the one guilty of the thrombosis. The delay because of this may prove very detrimental, as in Case 1 of this series. There we had a double mastoiditis, each side showing a perisinus abscess. It was two days after the patient had his initial chill before we were able to pin the diagnosis of sinus thrombosis upon the left side. The picture may be very atypical, with an insufficient number of the classical signs present to make a diagnosis. We may have to wait for the subsequent development of these signs to confirm what we may have already strongly suspected. Case 7 illustrates this. We may be confronted by the presence of some coincident disease which also gives a septic picture. Pulmonary complications or empyema may detract our attention from the sinus.

As soon as a diagnosis of sinus infection is made, operative interference should be resorted to. The earlier the operation follows the involvement of the vein, the more the patient will be spared. We are usually handicapped by difficulty in making a diagnosis and pay for the delay in a stormy postoperative course, or worse. Dench[12] considered that even in the absence of tenderness in the neck and other local signs, with an increasing remittent temperature and an almost certain knowledge of an infective sinus, it was wrong to delay operative interference.

If we must wait for these local signs, we are bound to have a prolonged course of sepsis. The difficulty is that we operate for the most part on signs caused by the general septic condition. If we can make our diagnosis upon its initial appearance and operate immediately we will gain a great deal. For instance, it is a mistake to wait for the appearance of chills if we have other sufficient data to proceed upon.

Authorities differ as to the proper course of treatment. Since Ballance first advocated ligation there have been differences of opinion as to the correct surgical procedure. At present there seems to be a division of opinion between ligation of the vein and complete resection. The operative work on the sinus proper is pretty much the same for both groups. The same method of procedure in regard to the vein may not be applicable to every case. McKernon,[9] Dench[12] and Richards[13] rather tend to resection, at least where there is any particle of doubt. On the other hand, the simple ligation can be performed in a very few minutes, causes very little trauma and very little shock to the patient, as compared to the more radical method. Tobey,[14] in a series of over two hundred cases, has found it necessary to employ resection in only two of them. If the horizontal crease incision is used the later cosmetic results are very gratifying. On the cases in this series on which this incision was used the neck scars were hardly discernible a few months afterwards. However, as Sauer[15] has said, after discussing the various methods of procedure, the results do not depend on any certain method, but upon how early the case is operated upon. For the ligation to prove satisfactory, in comparison to resection, it must be done early. In such cases it seems almost ideal. The ligation should be performed before there is any interference with the contents of the sinus, with the chance of sending infected material down the vein. If the mastoid has already been operated upon and we feel absolutely certain of the diagnosis—that the picture is not simulated by a perisinus or extradural abscess—we may start the operation in the neck with a perfectly fresh field and then after the jugular has been ligated and the wound sutured proceed to the necessarily infected mastoid region and the sinus.

In the series here reported all were operated upon as soon as we could establish the diagnosis. However, looking backward, it would seem that sinus operation was unduly delayed in almost every case. There is no doubt that each case would have done much better with earlier interference. This is a case of where "hindsight is better than foresight." Possibly, at the time some of these cases were operated upon, resection would have been preferable to the course pursued. It is a debatable point. Considering the condition at the time and the amount of shock to the patient, the simpler method seemed the better. It would seem that the mistakes made were really on the time of operating and not so much on the method.

The operation performed in all, except Cases 4 and 7, was the ligation, following the method of Dr. G. L. Tobey. With the exception of Nos. 1 and 2, they were all performed under nitrous oxid and oxygen anesthesia with most gratifying results. And right here it is not inappropriate to mention the very satisfactory use of this anesthetic. Shock was reduced to the minimum and the patient recovered consciousness before the final dressings were applied. In such cases as these, this was of great importance. Truly it seems the anesthesia par excellence for this type of case.

After usual preparation of the field, the head was turned toward the opposite shoulder and a sandbag placed under the neck to put it on the stretch. A small horizontal incision, not over one and a half inches long, was made in a deep crease of the neck in the superior carotid triangle, at the level of the thyroid, just anterior to the border of the sternomastoid muscle. The incision was carried through the platysma, retractors were introduced, Mayo scissors were substituted for the scalpel, and dissection made vertically just anterior to the sternomastoid. The carotid sheath was exposed, entered, and the internal jugular vein isolated from the carotid and the pneumogastric nerve. A double ligature of chromic catgut was placed around the vein, separated about a half inch and tied and the wound closed. Now the mastoid wound was reopened and the sinus widely uncovered. The upper and lower ends were compressed by gauze plugs inserted between the vein and the inner table. The sinus was incised, con-

tents evacuated, and a large portion of the external wall removed, to a point beyond the thrombus. After free bleeding had been obtained from above and below, the sinus was packed with iodoform gauze and the wound partially closed with silkworm gut. The gauze in the sinus was left, on an average of five days. The horizontal crease incision gives ample exposure if properly utilized and with proper retraction. The cosmetic result afterwards, while of secondary importance to conserving life, should not be overlooked. In Case 4 the operation was the same except for the neck incision, which was considerably longer and vertical, parallel to the anterior border of the sternomastoid. Case 7 showed considerable swelling and inflammatory reaction in the neck, and the long vertical incision was required. This inflammatory mass was removed and the vein resected for a considerable extent.

In the series here reported Case 4 was Dr. W. E. Sauer's and his courtesy is incorporated herein. In Cases 5 and 7 the initial operative work was performed by other members of the hospital staff, and the sinus operation and later care were by the writer. Cases 1, 2, 3 and 6 were the writer's throughout.

Case 1.—The patient was admitted to the hospital April 3, 1918, with a diagnosis of measles. He was a young white man, twenty-one years old, of slight build. Complicating his measles was a severe bronchitis. Eight days after entrance he developed an acute suppurative otitis media on the right. The membrana tympani was incised under local anesthesia. Two days later he developed a similar condition on the left. An incision was early performed. Both ears gradually quieted down and seemed to be convalescent in a few days.

April 25th. There was exacerbation of the otitis media on the right side requiring another incision. X-ray examination at this time showed large pneumatic mastoids, the cells apparently clear. From this time on there was considerable discharge from both ears, but otherwise than this the patient seemed to be very well and was able to be out of bed.

May 11th. There was increased mucopurulent discharge from the left middle ear.

May 12th. There was profuse discharge from the left ear, the superoposterior canal wall was slightly flattened, the mem-

brana tympani red and thickened, and there was slight mastoid tenderness and edema. The white blood count at this time was 11,800. Differential: small mononuclears, 27; large mononuclears, 5; transitional, 1; polymorphonuclears, 68.

May 12th. Operation. Left simple mastoid. Ether anesthesia. Usual incision—cortex bleeding—large pneumatic mastoid with free pus and considerable destruction of the cell walls, disclosing a perisius abscess. The mastoid was thoroughly exenterated and the antrum opened, lightly packed with iodoform gauze and the wound partially closed with silkworm gut. He reacted well from the operation and seemed somewhat improved the next day, but the following evening his temperature rose to 103.8°.

May 14th. He had a chill. The wound was clean, the middle ear dry, and there was no edema over the region of the emissary vein nor was there any tenderness in the neck. His white blood count was 9,200. The right ear showed a profuse mucopurulent discharge, and there was slight flattening of the superior canal wall. There was no mastoid tenderness. While we felt certain that this was a case of sinus thrombosis we did not dare to consider the left sinus as the affected one without exploration on the right.

May 14th. Operation. Right simple mastoid. Ether anesthesia. Usual incision—cortex bleeding—free pus and cell walls considerably broken down—perisinus abscess similar to the left. Mastoid exenterated, wound lightly packed with iodoform gauze, partially closed with silkworm gut. We were unable at this point to decide which sinus was affected.

May 15th. Temperature was 105°. White blood count, 12,800. The next day the white blood count was 17,600. Differential count: small mononuclears, 1; large mononuclears, 6; polymorphonuclears, 93. Packing was removed from the left wound and a small amount of pus seen on the sinus wall, which was pulsating. The right wound was clean and the middle ear about dry. It was deemed necessary to interfere and the evidence pointed toward the left side.

May 16th. Operation. Ligation of left internal jugular vein. Ether anesthesia. The internal jugular was isolated and ligated in the method above mentioned. The sinus was uncovered and incised. A good sized thrombus was found

and removed. A portion of the anterior wall was removed, the sinus curetted and iodoform gauze introduced.

Report of blood culture after forty-eight hours' growth was negative. Two days later a metastatic involvement of a metacarpal joint was noted. Packing was removed from the right mastoid wound.

May 20th. Temperature 103°; general condition seemed good.

May 21st. Medical consultation resulted in a diagnosis of bilateral bronchopneumonia. Blood culture still showed no growth.

May 24th. Both middle ears were dry and the wounds clean.

May 27th. Left pleural cavity aspirated and streptococci found in culture from the fluid. Medical consultant advised use of antistreptococcic serum. Considerable reaction followed its administration. After desensitization, 100 cc. of antistreptococcic serum with 150 cc. saline was given intravenously. This was repeated the next day. There was marked reaction following the use of the serum. The left arm became greatly swollen and cyanotic, with purpuric areas reaching to the shoulder. While the neck and mastoid wounds were perfectly clean and the middle ear was dry, patient seemed extremely sick. The temperature was about 103° each evening, and he was the picture of general sepsis. Further doses of antistreptococcic serum were given May 29th, 30th and 31st.

May 31st. Three hundred cc. of straw colored fluid removed from left pleural cavity. The temperature that evening was 99°. His general condition was very weak and stimulation was required.

June 1st. Five hundred and ten cc. turbid straw colored fluid removed from the left pleural cavity.

June 2d. Fifteen cc. antistreptococcic given intravenously. This was discontinued, as patient became very much distressed and his respiration and pulse very rapid during administration.

June 6th. He complained of pain in his left knee, and examination showed the presence of fluid in the joint. The same day 35 cc. of turbid straw colored fluid was removed

from left pleural cavity. The patient gradually became weaker—cerebration was impaired and he died on June 13th.

No autopsy was obtained. The factors in this case seemed to be a markedly virulent infection, plus the nonresistance of the individual, worn down by his long illness. Added to this we have the delay of two days in ligating the jugular because of inability to diagnose the affected side. This undoubtedly was of extreme importance. Also the use of antistreptococcic serum undoubtedly was of no benefit but, on the contrary, it seemed to us to have had a markedly detrimental effect. Each dose seemed to lower his resistance.

Case 2.*—Patient was admitted to the hospital April 20, 1918, complaining of pain in the right ear. Age, twenty-one years. Family history negative. Past history: mumps, measles and whooping cough in childhood; no previous ear trouble. He had had severe pain in the right ear, following a severe cold, for two days preceding admission. Examination showed a right suppurative otitis media. There was no mastoid edema, but he was quite tender over the tip. Membrana tympani was immediately incised, resulting in a serosanguinous discharge and relief of pain. Tenderness over the mastoid persisted in decreasing degree for two days and then subsided. On the tenth day the ear was dry. On the twelfth day the temperature rose to 104°. The ear was apparently convalescent. Examination of the throat showed an acute follicular tonsillitis. This subsided in about four days. On the nineteenth day after admission, he developed an exacerbation of his otitis media requiring a second incision. Because of quarantine of the ward on account of scralet fever it was impossible to have a radiograph taken at this time.

On May 10th, twenty days after admission, he developed a temperature of 103°. The ear was discharging freely, there was no mastoid tenderness or edema, but considerable preauricular swelling. Following a consultation with the medical service, patient was put under observation for mumps. Next day's temperature was 100.°

May 12th, twenty-two days after onset, there was profuse discharge from the middle ear, and the superior canal wall

*Abstract from report of case in Annals of Otology, Rhinology and Laryngology, Vol. XXVIII, No. 1, March, 1919.

showed a distinct flattening. The membrana tympani was red and bulging, the mastoid periosteum felt thickened, and there was slight tenderness over the tip. Temperature, 102.2°. Operation immediately performed. Ether anesthsia. Usual incision—cortex bleeding—large pneumatic mastoid with cells broken down and containing free pus, perisinus abscess; antrum opened and exenterated; iodoform gauze inserted and wound partially closed with silkworm gut.

Patient followed an uneventful course for four days, when he had a severe chill, following which the temperature went to 104. Packing was removed from the mastoid, and the sinus seemed to be pulsating. White blood count, 6,400. Differential: polymorphonuclears, 93; small mononuclears, 3; large mononuclears, 2; transitional, 2. Blood culture showed no growth. Next day patient seemed to be better, middle ear was dry and there were no chills. Temperature that evening, 102°; white blood count, 9,600.

May 18th, six days after operation, temperature was normal in the morning and the patient felt fine. At 4 p. m. he had a spontaneous hemorrhage from the lateral sinus, which was easily controlled by packing. Temperature that evening, 101.6°. Next day patient had rather a septic look, and temperature rose to 104.4°. White blood count, 16,800. Differential; polymorphonuclears, 72; small mononuclears, 11; large mononuclears, 7.

May 20th. Patient had a second chill, following which the temperature went to 103°. White blood count, 13,000. Differential: Polymorphonuclears, 85; small mononuclears, 5; large mononuclears, 10. Operation immediately performed under ether anesthesia. Usual ligation of the internal jugular vein and drainage of the sinus. No obliterating thrombus was found. Patient reacted well from operation.

Patient ran a septic course for about three weeks. He had some nausea and vomiting and several subsequent chills. The area under the neck incision became swollen and fluctuant, necessitating the reopening of the wound. Considerable pus was evacuated. We felt this was due to infection from a buried catgut suture, but it may have been a result of the phlebitis.

May 25th. Blood culture showed a growth of streptococcus hemolyticus.

May 26th. He complained of pain in the right shoulder joint. The next day there was another spontaneous hemorrhage from the sinus, easily controlled by packing.

June 2d. There was a third hemorrhage, but less in degree. He still complained of pain in the right shoulder joint and on June 6th 8 cc. of serosanguinous fluid was aspirated from the same. There was no growth from this on culture.

June 16th. Fundi were examined by Lieut. Col. de Schweinitz. "Right disc swollen 2.5 D., hemorrhage above disc. Left disc swollen 3.5 D. Probably toxic condition."

From this time on the patient's course was uneventful. The temperature remained normal and he was up and about. Subsequent examination of the fundi showed a gradual subsiding of the discs.

July 8th. Vision both eyes, 20/30. Hearing normal. This case agrees with the pathology of sinus thrombosis as described by Uffenorde.[16] The hemorrhages resulted from necrosis of the vessel wall. Lewis[17] describes a somewhat similar case. If ligation had been performed at the time of the first hemorrhage instead of waiting for the subsequent chills, undoubtedly the patient would have been spared a great deal.

Case 3.—Patient was admitted to the hospital January 29, 1919. Age, twenty-six years. Former occupation, farmer. Family history, negative. Past history: measles, mumps and whooping cough in childhood, typhoid fever ten years ago, lobar pneumonia nine years ago, influenza October, 1918, since which time he had been suffering from a lung condition which had been diagnosed as chronic tuberculosis. He was admitted to the hospital from a hospital train, en route to a tuberculosis hospital, complaining of pain in his right ear. Diagnosis: right acute suppurative otitis media. He was poorly developed and ill nourished and seemed ill and very weak. His right ear had been discharging one day. Examination of his lungs showed râles, diagnosed by the medical service as tubercular. There was a profuse discharge from the middle ear, the canal was negative and the membrana tympani red and bulging with a round central perforation. There was some tenderness over the tip of the mastoid. Membrana tym-

pani incised. The next day the tenderness had disappeared. He ran an uneventful course for the next seven days, when the discharge became increased in amount, the landmarks of the middle ear became lost, and the mastoid again became tender. The canal remained negative. X-ray examination resulted in a very poor plate. The cell walls were invisible, and it was reported as resembling a sclerotic mastoid. Temperature was normal. Diagnosis of acute mastoiditis made.

January 18th. Operation. Right simple mastoid. Nitrous oxid and oxygen anesthesia. Usual incision; thick cortex, rather small diploetic mastoid, partially broken down and with free pus. Perisinus abscess. Sinus found forward just behind antrum and showing granulations on its wall. Antrum well opened and mastoid exenterated. Iodoform gauze inserted in cavity—wound partially closed with silkworm gut.

January 20th. Temperature rose to 104.2 in the afternoon. White blood count, 22,000. Polymorphonuclear leucocytes, 81. No chills. Slight discharge from the middle ear. Wound clean.

January 21st. Temperature, 102° a. m., 102.6° p. m.

January 22d. Temperature, 102° a. m., 103.6° p. m. White blood count, 24,000. Polymorphonuclear leucocytes, 82. There were no chills, no localizing signs in the neck, or edema over the emissary vein. There was slight discharge from the middle ear. The picture was complicated by the obvious pulmonary condition. Although repeated consultations were held it was impossible to get a positive medical opinion as to whether the lungs were responsible for the temperature or not. Examination of the fundi was negaive. White blood count January 23d showed a decrease of 10,000. Polymorphonuclear leucocytes, 79. Blood cultures negative.

He continued a septic course until January 25th, when he had a chill, following which his temperature ascended to 104.4°. White blood count still 10,000. Polymorphonuclear leucocytes, 68. Report of blood culture after forty-eight hours' growth showed streptococci. At this time we were more or less embarrassed by delay in obtaining laboratory reports promptly. Diagnosis of sinus thrombosis made. Operation immediately performed.

Operation: Nitrous oxid and oxygen anesthesia. Internal

jugular vein ligated. Mastoid reopened and sinus widely exposed. A small button of exuberant granulations was seen on its anterior wall. Sinus opened and a portion of wall excised. No obliteration thrombus found.

The patient ran the usual septic course for the next seven days, during which time several intravenous salines were given in an effort to combat the infection already present in his blood stream. January 28th. White blood count, 22,000.

February 2d. There was an area of redness and puffy swelling with line of demarcation on the right side of his face. This was diagnosed as erysipelas, and he was put in the isolation ward where he ran the usual course of this infection.

February 7th. His temperature returned to normal, and his subsequent course was one of uneventful convalescence. Discharged from the hospital April 25th with his general condition greatly improved.

The factors in this case are the low resistance in a tubercular individual and the delay in ligating the jugular. The condition of the lungs and the possibility of their accounting for the sepsis due to the sinus infection held up our diagnosis. Earlier ligation before the advent of the chills would have been beneficial.

Case 4.—The patient was a young man, aged twenty-four years. Admitted to the hospital October 12, 1918, with a diagnosis of bilateral acute suppurative otitis media. Family history negative. Past history, usual diseases of childhood and a right otitis media at the age of five. Present history: Five days before admission had pain in both ears, followed by discharge the next day. Examination of the ears on admission showed slight thin discharge from both middle ears and canals negative. Membrana tympani red and bulging with a small round perforation in each. Both membrana tympani were incised to insure better drainage.

October 16th. A second incision of the right drum was required.

October 17th. Right superior canal wall was flattened, the drum boggy and discharge pulsating; mastoid area showed slight edema and some tenderness; similar condition on the

left side. Operation: Double simple mastoid. Nitrous oxid and oxygen anesthesia. Thick cortex on both sides, both antra filled with pus; in the right there was some pus in the cells but none on the left. Uneventful course until October 28th, eleven days after operation, when his temperature rose to 103°. Both middle ears were dry at this time and the wounds were clean.

October 30th. Complained of some stiffness of the muscles of the neck, but there was no rigidity. Temperature had returned to normal. Examination of the fundi showed slight hyperemia of both discs.

November 3d. There was slight discharge from the right ear.

November 5th. Temperature rose to 102°, and patient complained of being chilly—no evidence of definite septic chill. Reflexes negative. Slightly tender over right jugular vein. White blood count, 14,500. Differential: Polymorphonuclears, 76; small mononuclears, 20; large, 2; transitional, 2.

November 7th. Complained of pain in right zygomatic region.

November 8th. Temperature, 103°; pulse, 102. Lumbar puncture performed; fluid clear and under very slight pressure; examination of fluid negative. Neurologic examination negative. Patient ran a slightly septic course for the next three days.

November 10th. Fundus examination disclosed hyperemia of the discs. the same as in previous examination, but considerably more venous engorgement. Area over right jugular vein quite tender. White blood count, 17,000; polymorphonuclears, 86; small, 6; large, 8. Blood culture showed no growth. Diagnosis of sinus thrombosis was based upon the patient's septic appearance, temperature and the tenderness in the neck and fundus examination.

November 10th. Operation. Nitrous oxid and oxygen anesthesia. Mastoid wound reopened and granulations removed from cavity; sinus exposed from the knee to the bulb; wall appeared necrotic at lower portion and pus was seen coming from the region of the jugular fossa. Wound temperately packed with iodoform gauze and internal jugular vein ligated. Sinus was then incised and a portion of external wall removed,

but no obliterating thrombus found. Sinus was packed with iodoform gauze after free bleeding had been established from above and below, and the wound patrially closed with silkworm gut.

Patient seemed to be somewhat better the next day. Temperature continued high at night for about five days, after which it gradually came down to normal.

November 13th. Area of swelling was noted just below the mastoid tip, at the angle of the jaw, which subsided in three days.

November 21st. Swelling noted again, but this time farther downward in the region of the jugular incision. Next day a distinct fluctuation could be made out over this area, and the incision was reopened under nitrous oxid and oxygen anesthesia, and a considerable quantity of pus evacuated, just anterior to the sternomastoid muscle. Iodoform gauze drain. This was removed in four days and the wound was allowed to heal.

November 24th. Examination of fundi showed the discs less hazy, vessels about normal in size. The right mastoid wound was healed December 22d; left, December 31st, and the patient restored to duty January 2, 1919.

One of the main factors of this case was the virulence of the infection. No perisinus abscess was found at the time of the mastoid operation, the plate covering the sinus apparently being healthy. At the second operation this was found softened with apparent necrosis of the vessel wall. The absence of well defined chills and the negative blood culture is of interest. The patient undoubtedly got his initial sinus infection November 5th, but delay in making the diagnosis postponed interference five days.

Case 5.—Patient was a young white man, age twenty-one. Admitted to the hospital September 2, 1918, with a diagnosis of acute pansinusitis. Had been having headache and profuse purulent discharge from both sides of the nose for three weeks previous to admission. X-ray examination showed slight cloudiness of all sinuses. Patient was put upon treatments of hot irrigations.

September 8th. Six days after entering hospital patient complained of pain in the left ear; membrana tympani was

found to be red and bulging. Incised under local anesthesia, resulting in a serosanguinous discharge. There was slight tenderness over the tip. Culture from the ear showed bacillus diphtheriæ. Nasal condition had subsided.

September 8th. X-ray examination showed distinct cloudiness of the left mastoid.

September 11th. Rather profuse discharge; superior canal wall slightly flattened; some tenderness over the mastoid. Operation. Left simple mastoid. Nitrous oxid and oxygen anesthesia. Notes of this operation could not be obtained, so conditions found at operation were unknown to us. Uneventful course until November 22d, during which time cultures became negative for diphtheria.

November 22d. Developed a temperature of 103° rectal. Middle ear and mastoid wound were discharging profusely. There was some tenderness over the zygoma. Temperature normal the next day. Tenderness over zygoma persisted, and on November 20th superior canal wall appeared flattened and the wound was discharging profusely.

November 22d. Temperature again rose to 105° rectal. General examination was negative. There were no chills and no tenderness in the neck. Reflexes all negative. There was a slight spontaneous rotatory first degree nystagmus to the right. White blood count, 6,650; fundi negative.

November 23d. Condition shows no change; white blood count, 7,000.

November 25th. Temperature, 105.2; some nausea and vomiting; no chills; reflexes negative. Lumbar puncture performed; fluid clear; no increase of pressure; cell count normal; globulin negative. Nystagmus had disappeared.

November 26th. Report on blood culture positive for streptococci. Operation. Nitrous oxid and oxygen anesthesia; mastoid wound reopened and granulations removed from cavity. Good sized area of dura found exposed. Considerable number of zygomatic cells containing pus were found and cleaned out; some remaining tip cells cleaned out; plate over sinus necrotic and was removed; sinus wall appeared thickl ened and rather gray. In the angle between the middle fossa and the sinus the curette opened up a large pus pocket. Wound was packed with iodoform gauze and partially closed with

silkworm gut. The next day the patient seemed better and the temperature was down.

November 29th. Temperature rose again to 104°. There were no chills. White blood count, 7,000. Reflexes negative. Middle ear was dry and the wound clean. There was considerable tenderness in the neck along the jugular vein. Operation, nitrous oxid and oxygen anesthesia. The internal jugular vein was ligated in the manner before mentioned and the wound closed with silkworm gut. Mastoid wound reopened and sinus incised and good sized thrombus removed. A portion of anterior wall was excised and the sinus packed with iodoform gauze; wound partially closed. Patient ran a septic course for the next twenty days.

December 3d. He has his first chill.

December 4th. Packing was removed from the sinus and pus was seen coming from the lower portion. Iodoform strip inserted toward the bulb. This was changed daily and, after two days, no pus was noted.

December 12th. Second chill, following which temperature went to 104.6°. Some nausea and vomiting. Wound clean. No nystagmus. Cochlear and labyrinth apparently not involved. Reflexes negative.

December 18th. Temperature normal and convalescence was uneventful from this time on. Restored to duty January 26, 1919.

The main factors of this case seem to be incomplete mastoid operation in the first place, plus a lack of resistance on the part of the patient as shown by the absence of any leucocytosis. Operation on the sinus was undoubtedly delayed. It should have been performed at the time of the secondary mastoid operation. It is to be feared that lack of confidence in the laboratory finding of a streptococcic blood culture had a great deal to do with this delay. It is easy to see now that interference on the sinus would have been the proper course to have pursued, but at that time we hesitated to convict the vessels on a septic temperature and positive blood culture, without first cleaning out the infected mastoid area. This is another case where more confidence and cooperation among the different branches of the profession would have reacted to the patient's good.

Libman says: "If the middle ear has been properly drained and the mastoid operated upon, and there is no meningitis or other cause found to account for the bacteremia and persistent symptoms, by exclusion, we must assume a lesion present in the veins."

The mastoid area was undoubtedly incompletely cleaned out at the first operation, and it was proper for us to rule this out first. However, the appearance of the sinus at the second operation was very suggestive of a thrombosis. Looking backward on the case now, there seems to be no doubt that the sinus should have been opened at this time and the jugular ligated, another error on the part of conservatism. The diphtheritic otitis, at the onset, apparently had little significance as regards the subsequent sinus involvement.

Case 6. Patient was a well developed man of thirty-eight years. Admitted to the hospital January 24, 1919, with a diagnosis of right acute suppurative otitis media. Family history negative. Past history negative. On admission, membrana tympani was incised, resulting in a slight thin discharge. The ear improved until January 30th, when because of increased and pulsating discharge it was necessary to incise the membrana tympani again.

February 1st. There was a slight flattening of the superior canal wall, but no mastoid tenderness or edema.

February 2d. Temperature, 101.4° p. m.

February 3d. Operation. Right simple mastoid. Nitrous oxid and oxygen anesthesia. Usual incision; cortex extremely thick and mastoid found broken down and cavity filled with free pus. There was a perisinus abscess, sinus far forward and covered with granulations. The mastoid was exenterated and the cavity filled with iodoform gauze and wound partially closed with silkworm gut.

February 4th. Temperature, 101.6° a. m., 103.8° p. m. This gradually came down until February 8th, when it was 98° a. m., 100.4° p. m. The next day temperature rose to 105° in the evening. Evening temperature continued between 104° and 105° for the next two days, with a slight discharge from the middle ear. Wound was clean. There were no chills. Examination of the lungs showed transient râles at both bases.

February 9th. Examination of the fundi showed slight blurring on the nasal side. Reflexes were negative.

February 10th. Middle ear was dry. Temperature gradually came down again until February 15th, when he had a chill, following which temperature rose to 105°. Examination of the urine showed some pus present.

February 15th. He had another chill in the evening. White blood count, 13,400; polymorphonuclear leucocytes, 77. Blood culture negative. Diagnosis of sinus thrombosis made.

February 16th. Patient had another chill before operation could be performed. Operation. Nitrous oxid and oxygen anesthesia. Usual ligation of internal jugular vein and an exploration of sinus revealing a parietal thrombus; anterior wall excised and sinus packed with iodoform gauze. Ran a septic course until February 25th, when temperature was 99° a. m., 100° p. m.

February 19th. After being typed, patient was given a blood transfusion. Neck wound was healed, middle ear dry and mastoid clean. Blood cultures persistently negative.

February 26th. Complained of pain in right elbow.

February 27th. Temperature rose to 105 coming down to normal the next day. There was another rise of temperature March 3d following a chill. Septic course for the next five days. Swelling noted in neck under old healed incision. This was reopened and considerable pus evacuated. Examination of fundi normal. Uneventful course from now on. Elbow responded to simple treatment.

The essential factors in this case were the virulence of the infection and the delay in operation because of the thick cortex masking the signs. The pus in the urine distracted our attention from the sinus for a time, and the absence of chills, positive blood culture and the slight leucocytosis delayed operative interference upon the sinus too long for the patient's good.

Case 7.—The patient was a young white man, age twenty-two. Admitted to the hospital December 29, 1918. Diagnosis, measles. Family history negative. Past history, mumps and whooping cough in childhood, typhoid fever nine years ago, appendectomy two years ago.

January 5th. Complained of pain in the right ear. Examination showed a right acute suppuraive otitis media. Membrana tympani incised, resulting in serous discharge. Next day there was slight mastoid tenderness.

January 7th. The tenderness was increased, and there was a profuse discharge from the middle ear, with complete loss of landmarks. Temperature was 103°.

January 8th. Condition the same. Temperature, 102°.

January 9th. Profuse discharge from the middle ear. Increased mastoid tenderness. Temperature, 103°. Operation. Right simple mastoid. Nitrous oxid and oxygen anesthesia. Usual incision showed large pneumatic mastoid, and cell walls not broken down, but containing free pus. Antrum opened and mastoid exenterated. Iodoform gauze to cavity. Wound partially closed with silkworm gut.

January 10th. Temperature, 101° a. m., 102.2° p. m. This gradually came down to normal on January 14th. The mastoid wound was clean but the ear was discharging. This condition persisted until February 5th, when he complained of headache and stiff neck. Temperature was normal, pulse 72. There was a question of rigidity of the neck and a suspicion of a positive Kernig. White blood count, 19,300. Considered to be meningeal irritation but neurologic examination resulted in a diagnosis of psychoneurosis. Lumbar puncture was performed. The fluid was clear and under no increased pressure, cell count, etc., were normal. Fundi negative. Heart and lungs negative. Urine negative.

February 6th. It was noted that there was a paralysis of the right external rectus muscle. Temperature, 100.4° p. m. Cochlea and labyrinth responded normally to tests. No involvement of fifth nerve. Middle ear still discharging, and there were unhealthy appearing granulations in the wound. No change for the next four days. Secondary operation advised.

February 10th. Operation. Secondary mastoid. Nitrous oxid and oxygen anesthesia. Wound reopened and granulations cleaned out. Epidural and perisinus abscess found. Sinus apparently patent, although gray in color. Iodoform to cavity. Wound partially closed.

February 11th. The canal appeared swollen and tender and there was considerable discharge.

February 14. The right shoulder joint sore. Nothing found on examination. At this time patient was running an afternoon temperature each day of about 101°. Blood cultures persistently negative.

February 19th. White blood count, 10,000. Wassermann test negative.

No change until February 27th, when a granulating fistula was noted in the floor of the canal which had been swollen for some time. A probe was passed for a considerable distance downward toward the floor of the middle ear. Apparently the middle ear was dry and the discharge coming from this tract. There was slight swelling noted in the neck just below the tip of the mastoid.

February 28th. Swelling in the neck had increased. This was tender but not fluctuant. It seemed to be rather a brawny induration. This gradually increased downwards until it reached the level of the thyroid. Patient was running a temperature of about 101° every afternoon and was the picture of a low grade sepsis. Heart, lungs and urien negative.

This was considered probably a case of atypical sinus involvement. The continued, if low grade, sepsis following one rise of temperature to 103°, and the presence of localizing signs in the right side of the neck made exploration at least advisable.

March 6th. Operation. Nitrous oxid and oxygen anesthesia. Incision of neck parallel to the anterior border of the sternomastoid. Deep dissection revealed a large inflammatory mass, including large lymphatic glands, the whole being adherent to the carotid sheath. This mass was dissected out, the sheath opened and the internal jugular vein found to be collapsed. This was ligated and resected for a considerable distance. The mastoid was reopened and granulations removed from the cavity. The plate covering the sinus was very soft and necrotic in the lower portion, where it remained from previous operation. Sinus exposed downward almost to bulb and wall found covered with granulations. Sinus opened and found thrombosed. A portion of the anterior wall was removed and after being curetted to the bulb the sinus was

packed with iodoform gauze and the wound partially closed with silkworm gut. The neck wound was drained for three days and then allowed to heal.

Following the operation the middle ear was dry, and there was no more discharge from the canal. The swelling gradually subsided in the neck and the canal opened up. The temperature continued as before for a few days, but after March 15 it remained normal. The mastoid wound was healed by March 24. The paralysis of the sixth nerve subsided in about two weeks. Restored to duty April 1.

Here we had almost a fulminating mastoiditis following measles, complicated by rather an atypical sinus infection. So rarely does a case follow the classical textbook picture that it is almost to be doubted whether there is any such thing as a typical or atypical form. This might follow the type Tobey[14] speaks of as typhoidal. The initial invasion of the sinus probably took place January 10, with the rise of temperature to 102.2°. Following this we had a long continued low grade sepsis. There were no chills. Blood cultures were persistently negative. After February 5 there was no marked leucocytosis. Whether the sixth nerve paralysis was due to infection of the inferior petrosal sinus from the bulb, as in the cases reported by Pierce,[18] or was purely toxic, it is difficult to say. This is not infrequently seen in acute suppurative otitis media. The meningeal irritation was not something absolutely uncommon. Serous meningitis has been seen in sinus thrombosis. He was watched for the possibility of Gradenigo's syndrome, but no involvement of the fifth nerve was ever noted. Apparently the bulb was involved. In the case reported by McCoy,[19] the suppuration in the bulb persisted, requiring the performance of the Tandler operation. This case responded rather promptly to simpler measures, curettage and drainage of the bulb with removal of infected material from the neck.

ETIOLOGIC FACTORS.

Virulence.—The virulence of the infection seemed to play an important rôle in all the cases of this series except possibly cases 3, 5 and 7. In Case 7 it manifested itself by the acute fulminating type of mastoiditis, after which this factor seemed to lose its prominence and the case ran quite an atypical course.

Low Resistance.—This factor was quite evident in Case 1, where the patient was anything but rugged to start with, and in addition was considerably worn down by measles and bronchitis. On the contrary, Case 2 showed remarkable resisting powers, which enabled him to overcome his infection. Case 3 was tubercular and showed low resistance both in a general way, and in the low white count seen shortly after the onset of his sinus infection. Case 5 likewise exhibited poor resistance and never showed any increase in leucocytes during any stage of the disease. Cases 4, 6 and 7 showed good resisting power.

Delayed Operation.—Delay in performing the mastoid operation was an essential factor in Cases 1, 2 and 6. In the first, however, operation was performed on the side which was later thrombosed, about as soon as the picture warranted. Operation on the right side, a day or two earlier, might have helped in conserving his general resistance. Cases 2 and 6 were delayed because of masked signs. It is only fair to say that all the cases show a delay in performing the sinus operation, due to tardiness in making our diagnoses. Frequently we lack the courage of our convictions. This series seems to point out this common error in an unmistakable way.

Incomplete Operation.—Two cases, Nos. 5 and 7, are examples of this. The mastoid was not thoroughly cleaned out at first operation in either case, and the pathologic process went on until there was an invasion of the sinus.

PATHOLOGIC FACTORS.

All the cases in this series, with the exception of No. 4, showed perisinus abscess, although in Cases 5 and 7 this was not noted at the first mastoid operation. However, these were the cases which later gave evidence of having been incompletely exenterated. At the second operation, a perisinus abscess was found in both cases. The process was the confluent, or cell route, variety. The invasion of the sinus in Case 4 was apparently by necrosis of the inner table and direct extension to the sinus wall.

The bacteria, as demonstrated by blood culture, was streptococcus in Cases 1, 2, 3 and 5. No organism was isolated in Cases 4, 6 and 7.

SIGNS AND SYMPTOMS—CLINICAL FACTORS.

Temperature.—As has been pointed out by McKernon, Dench and others, this is by far the most important symptom. Each case ran a typically septic temperature, except No. 7, in which there was one rise to 102.2°, followed by a slight rise of one or two degrees every afternoon. If it had been possible to rule out other febrile conditions, this alone should have enabled us to have made our diagnosis earlier.

Pulse and Respiration.—In all the cases pulse and respiration were proportionate with temperature in their deviation.

General Appearance.—Sooner or later all the cases showed the picture of general sepsis. However, it would have been decidedly advantageous to have made the diagnosis and instithe proper surgical course before the appearance of this sign. To interfere in time to minimize general sepsis should be our endeavor.

Chills.—All but Case 7 exhibited chills, although these were not seen in Case 5 until after ligation. Here again we have a symptom often appearing rather late in a disease, and it would seem a mistake to wait for its appearance, if possible to make a diagnosis without it. Malaria must be ruled out before accounting for the chills by sepsis.

LOCALIZING SIGNS.

Tenderness in the Neck.—This was noted in Cases 4, 5 and 7. In the last it, indeed, served as our guiding star, pointing out to us the proper course when other signs failed. When present it is decidedly helpful, but absent, it means nothing.

Edema Over the Emissary Vein.—This was noted only in Case 1. Like the preceding, it is useful only when present; indeed many consider this due to pus under the periosteum and not a sign of venous obstruction. The zygomatic edema and swelling seen in Case 2 was undoubtedly due to the involvement of the zygomatic cells and had no significance as regards the sinus.

Fundi.—Blurring of the disc margins with varying degrees of venous engorgement was noted in Cases 2, 5 and 6, but it was too late a factor to be of much diagnostic aid. Of these

three, Case 6 showed only slight changes. Subsequent examinations, during convalescence, showed the discs returning to normal.

Case 7 showed a paralysis of the right sixth nerve, probably either toxic or caused by extension of the process via the inferior petrosal sinus to the apex of the petrous.

Nausea and Vomiting.—This was noted in Cases 2 and 5, and then only in the later course of the disease, as an element of the general sepsis.

Leucocytosis.—Cases 1, 2, 4 and 6 showed a characteristic leucocytosis, with an increase of polymorphonuclear leucocytes of over 80. Ac McKernon has pointed out, this last is indicative of an infective condition demanding interference. Case 3 showed a typical leucocytosis and then a sudden drop to 10,000 just before operation was performed, probably due to a lowering of his resistance. Case 5 showed almost a leucopenia throughout, obviously a case of low resistance. Case 7 showed a slight increase in white cells. While the presence of a leucocytosis is of aid in making the diagnosis, it is mainly indicative of the patent's condition. The increase of polymorphonuclear leucocytes in the differential count is of more value.

Blood Culture.—Four of the cases, 1, 2, 3 and 5, gave a positive blood culture, although not always early enough to be of greatest diagnostic value. Three were persistently negative, although without doubt a bacteremia existed. According to Braun, coincident diseases in which it is possible to have a bacteremia are acute tonsillar infections, acute bacterial endocarditis, meningitis, scarlet fever, typhoid fever, pneumoina and possibly erysipelas. Ruling out these other conditions, a positive blood culture in a case of mastoiditis running a septic temperature, otherwise unaccounted for, should make the diagnosis of sinus thrombosis certain.

Metastasis.—Four of these, 1, 2, 6 and 7, showed metastatic involvements, but only in the last was this of diagnostic aid. This is really one of the end results of the disease and rarely occurs early enough to serve as a basis for diagnosis. Often it is confirmatory evidence.

SUMMARY.

Of the seven cases in this series, six showed a typically septic temperature, general septic appearance and chills, although occurring late in two of them. Two of these six cases showed localizing signs in the neck, and one other, edema over the emissary vein. Five showed a leucocytosis with increase of polymorphonuclear leucocytes, while one showed a leucopenia. Four showed positive blood cultures, all of them streptococcic. Three cases showed fundus changes. Metastases developed later in three of the six. One case showed one moderately high rise of temperature, followed by a daily elevation each afternoon of one or two degrees. This case also showed a slight leucocytosis, paralysis of the sixth nerve, and later inflammatory signs in the neck and metastatic involvement of the shoulder joint.

CONCLUSIONS.

1. The best prophylaxis for sinus thrombosis is thorough exenteration of the mastoid as soon as a diagnosis of suppurative (operative) mastoiditis is made.

2. Despite early and thorough surgery, sinus infection will occur in certain cases because of the virulence of the infection or the low resistance of the patient.

3. Operative interference should be instituted as soon after sinus infection has taken place as possible. Early operation will avoid much long drawn out and stormy postoperative period.

4. If performed early the ligation of the internal jugular vein, followed by exploration of the sinus and removal of its external wall, would seem sufficient. The horizontal crease incision leaves the best cosmetic result.

5. Temperature is the most important symptom. If otherwise unaccounted for, a septic temperature in a mastoid which has been thoroughly operated should be considered as indicative of sinus infection. Other conditions which might possibly cause a bacteremia being ruled out, a positive blood culture makes this diagnosis certain. Chills, the blood picture, localizing signs, etc., are important if present.

6. Operation should be performed as soon after the onset of the infection, as shown by the temperature, as it is possible to make a diagnosis.

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LXXVIII.

THE EDUCATION OF THE VESTIBULAR SENSE.*

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This study is restricted entirely to physiology. For the sake of clearness, certain definitions should first be made. The vestibular apparatus is defined as including the vestibular portion of the internal ears, the intracranial pathways from this portion of the internal ears, and the brain centers. The term, vestibular sense, is restricted to the function of this apparatus. The vestibular sense is one thing, "deep sense" another thing, eye sense another, and touch another.

Each organism has developed its own special sensory equipment to meet the particular needs of its life and environment. In order to avoid catastrophe and hazard in coming in contact with the external world, one of the first requisites of animal life is the sensing of motion. The lowest forms of animal life, like the coral, come into existence, live and die without changing position. As motion is no part of its life, the coral has no need for motion sensing. Higher in the developmental scale, animal life is freed from an anchored existence and is tossed about upon the ocean bed by the waves; motion becomes a natural part of its life destiny and motion sensing becomes a requisite. The primitive otic cup develops to fill this need; and sensory hairs are acted upon by water and sand particles. The amphibious animal, during its land excursions, becomes exposed to air borne wave shocks of greater delicacy and of more or less regular wave length. These cannot be sensed by the already developed motion sensing apparatus, and in response to this new need it develops a primitive cochlea. In animals living in fluid media the need of motion sensing is naturally greater; some fishes develop upon their surfaces additional sense organs, enabling them to sense all motion accurately and instinctively—

*Read before the American Otological Society, Boston, June 1, 1920.

the socalled linear endorgans. Thus their equipment includes visual sense, vestibular sense, deep sense and "visual line" sense. Similarly, in birds the semicircular canals are developed to a relatively enormous extent, in response to their particular necessity for full and instinctive possession of adequate knowledge concerning motion.

The blank cerebral cortex of the baby begins to receive messages from his special senses. That is the beginning—the end result is the fully developed child with normal equilibration. What are the steps of this development?

The human infant is endowed at birth with only four known sensory gifts which he may utilize for motion perception: vision, "deep sense," touch, and vestibular sense. These constitute the means of his acquiring information and experience with respect to motion; upon the proper use of such sensory information depends his further development of certain motor abilities. When he first attempts to sit up, the force of gravity pulls him over and he bumps his head—thereby encountering pain, perhaps for the first time. As he is being pulled over by gravity he sees he is falling, he experiences a sensation of falling coming from his deep sense and that coming from his ears. With repeated experiences of this sort, his memory gradually stores up the knowledge that, given this deep sense, visual and vestibular testimony, pain (the bump) is inevitable. His developing mentality commences to interpert these sensory impulses which tell him he is beginning to fall; appropriate motor coordination begins to develop and then become evident attempts to check the fall. Eventually he becomes able to sit up without falling. Still later, in the same way, he becomes able to stand, then to walk, then to run.

Whereas sight, deep sense and touch contribute to the general detection of motion, yet, for critical requirement, it was apparently necessary for all animals that move about to possess, in addition, an organ whose sole function is the sensing of motion. To fill this need, nature developed within the human bony cranium a single physical apparatus—the vestibular portion of the internal ear. Just as the eyeball is the prototype of the camera, and the tympanum the prototype of the telephone, so this apparatus is the prototype of the

seismoscope. The eyeball is devised for only one physical operation—the focusing of light waves; the tympanum, with its vibrating drumheads and ossicular chain, is devised for only one physical operation—the transmission of sound waves; just so the semicircular canals and the saccule and utricle with their fluid content bathing erect hairs, make up an instrument devised for only one physical operation—the detection of motion in every direction. The impulses from this motion sensing organ, integrated in the sensorium with those from all other sensory sources, go to make up the individual's composite motion perception. To what degree each one of the special senses contributes to perfect motion sensing is not known; we do know, however, that the absence of any one of these elements impairs full motion perception. Observation shows that the equilibration of any individual who lacks one of the three main fundamentals to motion sensing is below that of the normal. For example, the individual lacking vestibular sense, or the tabetic lacking deep sense, cannot stand on one foot with closed eyes. The reason for this inability is that his motion sensing composite is inadequate for necessary motor coordination; beginning motion of the fall is not sensed with sufficient speed and delicacy to permit him to make necessary motor adjustment in time to prevent the continuation of the fall. The normal "catches himself" before the falling becomes evident.

Studies of the vestibular sense are comparatively so new that it might be well to attempt to state what we do and do not know at the present time concerning the semicircular canals and their intracranial connections. When we all went to medical school the course in physiology included several weeks on the eye, several weeks on the organ of hearing, and considerable time on the study of the other special senses. Is it not rather striking to remember that all we heard in our physiology course was a casual mention, somewhat as follows: "The semicircular canals are situated posterior to the cochlea, and are 'concerned with equilibrium.' We will now take up the study of the heart."

Why was the vestibular sense overlooked through so many years? Perhaps because motion sensing is not so obtrusive in our everyday life as are sight, hearing, smell or taste—

those senses which minister to our pleasure or displeasure. Pleasant and unpleasant sights, beautiful music and harsh noises, agreeable and disagreeable odors, and the taste of good food or bad—these things obtrude themselves upon our notice; the perfect sensing of motion does not attract our attention. Here all the time was a good servitor who did not make his presence felt.

Another very practical reason for our comparative ignorance of the vestibular apparatus is that there were no technical methods devised for testing or studying this mechanism until the comparatively recent introduction of the turning, caloric and galvanic tests, whereas for a long time we have had the means at hand to test and examine the other sense organs.

In spite of the present limitations of our knowledge, is it not, however, a fact that there are certain things which, even now, we know to be true and accurate about this apparatus? We have a clear knowledge of the microscopic anatomy of the internal ear, due particularly to the work of the Vienna school and G. E. Shambaugh, and the physics (or mechanics) of the semicircular canals has been made clear. Further, it would seem that the following fourteen points are fairly established:

1. This end organ is developed phylogenetically and embryologically second of all—preceded only by touch.

2. The horizontal canals may functionate independently, whereas the vertical canals—the superior and the posterior—always act together for a mechanical reason—because of the existence of a common crus.

3. The horizontal canal is connected with the third and sixth pontile nuclei. The obvious proof is that stimulation of the normal horizontal canal always produces a horizontal pull of the eyes.

4. The vertical canals are connected with the third and fourth nuclei. Proof: Turning or caloric stimulation of the vertical canals produces vertical or rotary—but never a lateral—movement of the eyes.

5. The horizontal canal fibers and the vertical canals fibers have separate and distinct intracranial pathways after the eighth nerve enters the brain stem. (Pages 122 to 128, book "Equilibrium and Vertigo.")

6. Impulses producing nystagmus follow different nerve paths from those producing vertigo.

7. Stimulation of the ears produces only the slow pull of nystagmus, not the quick jerk.

8. The eyes are always pulled in the direction and plane of the endolymph movement.

9. In the normal, stimulation of any canal causes a sensation of motion.

10. This sensation of motion, vertigo, is always in the exact plane of the endolymph movement and in a direction opposite to the endolymph movement.

11. Past-pointing is not a "Reaktion" (Barany): it is a voluntary attempt to locate the external object at a point where it is not. Both past-pointing and falling are expressions of a sensation of motion.

12. The normal duration of after-turning nystagmus is 24 seconds.

13. Normal responses to ear stimulation determine definitely that the vestibular apparatus is intact.

14. Failure of normal responses to ear stimulation is definite evidence of some abnormality of the vestibular apparatus.

Having up to this point discussed the physiology of motion sensing, and having emphasized that the vestibular sense is only one of the four senses concerned, we might now take up examples of education in motion sensing. In this connection we might well ask ourselves, What is known about aviation, otologically, that we did not know three years ago? We know:

1. An individual can become an excellent flier without being perfectly normal physically. As Glen Curtis puts it, "Anybody who can ride a bicycle can fly an airplane." The "mystery" that surrounded aviation in the early days is now cleared up. Flying is a much simpler matter than we had thought.

2. While everyone can fly, safety demands that not everyone should fly. War experience has taught the other countries the necessity of raising their original standards of physical requirements. The man with good heart, lungs and kidneys, normal sight, normal vestibular sense and stable nervous sys-

tem is a far safer pilot than the man laboring under the handicap of any physical deficiency.

3. We now know that no set of tests or special examinations yet devised in any country can predict whether any given individual will become a good flier or not. This demonstrates the wisdom of the United States Air Service in never attempting any such prophecy. So many have considered that tests were useful only as they "correlated with flying ability." It cannot be done. The requirements of our service were the securing of a normal individual; this increased the factor of safety and made more sure that the flier's future usefulness would not be impaired by discoverable physical defect.

4. We now know that the ear plays a far greater rôle in flying than we originally suspected at this meeting three years ago. It is well enough to have a normal ear, but we have found that the brain must be trained to interpret the unusual impulses coming from the internal ear in aerial acrobatics.

Many crashes and deaths are directly traceable to this lack of education in interpreting the ear stimuli. We have learned that it is possible to educate the vestibular sense and also the visual and deep sense through actual aerial acrobatics; or more safely, by a course of training in an orientator. It must be further noted that an acquired education of the vestibular sense may be lost or partly impaired through disease. Hence a perfectly normal and trained flier may, through fatigue, toxemia or what not, become unfit for flying. Dangers from this source can be prevented by frequent re-examinations.

Taking up each of these points in further detail:

1. We now know that one eyed men, men with noticeably impaired semicircular canal function, one legged men—all may make good fliers; the immortal Guynemer accomplished his best work while dying from consumption—he had hemorrhages from the lungs from time to time and then would return to active flying duty.

2. Medical men of different specialties in all countries were asked to study the physiology of flying and to attempt to devise a series of tests which would eliminate the possibly unfit from their respective air services. Vestibular tests

were made an important feature of the examination in every country. The Germans and Austrians required the caloric as well as the turning tests of the ear mechanism; these tests were conducted by their most skilled labyrinthologists. The Italian Air Service work was under the direction of Gradenigo; special turning chairs were devised and a new method of making the pointing tests was used. The French used both the turning and caloric tests and employed specially constructed turning chairs. Belgian labyrinthology is best represented by the electric turning chair and nystagmograph of Buys of Brussels. The Canadians also routinely used the turning chair and in doubtful cases used the caloric test, as we did.

The English, like all the others, felt it important to determine that the vestibular mechanism was normal; their present point of view is perhaps best expressed by quoting "Reports of the Air Medical Investigation Committee on the Sense of Stability and Balance in the Air." Dr. Henry Head, chairman of the Investigation Committee, gives the following conclusions (page 43):

GENERAL SUMMARY.

"1. Conscious stability and comfort in the air depend primarily on the normal response of three groups of sense organs. These are the vestibular apparatus, the eyes, including the eye muscles, and the proprioceptive system with its endings in muscles, tendons and joints . . . We are interested in those impressions which lead to recognition of the position of the body in space.

"2. Defective responses from any one of these afferent end organs will disturb the sense of equilibrium in the air and may lead to actions that affect the stability of the machine.

"3. No candidate for service in the Royal Air Force is accepted who shows gross abnormality of any one of these primary organs of sensation."

This expresses in a very clear way the exact viewpoint of the United States Air Service also. During the war, however, the English method of determining "vestibular stability" did not include the turning or caloric tests; on reviewing the otologic work of the English, Dr. Head states: "'Vestibular stability' is estimated by the power to walk along a line, to turn on one foot and to stand on one leg with the

eyes closed. But there is no evidence to show that any of these acts bears a direct relation to the activity of the semicircular canals. Moreover, I have examined four pilots who were intensely giddy and vomited in the air, none of whom had the least difficulty in carrying out these tests perfectly. . . . There is nothing to show that inability to walk a line and to turn on one foot is evidence of anything more than clumsiness. . . . One cadet found difficulty in walking heel to toe and turning on one foot. He ultimately became an excellent scout pilot, but confessed that he disliked dancing. In fact, it seems to us that this test is more likely to show ability to dance than any capacity to fly. But whatever relation it may bear to flying, it is certainly not evidence of 'vestibular stability.' . . . Of the ten who had been 'giddy' and vomited, nine reacted excessively to rotation in the chair. . . . We can bear with impunity certain violent disturbances of equilibrium if the stimulus is expended on certain parts of the vestibular apparatus, but not if it falls on other combinations of the semicircular canals. . . . The majority of normal men do not suffer from these symptoms in the air. There are, however, some in whom the physiologic response is so violent that it can never be overcome, and they are always giddy and nauseated whenever there is any sudden or considerable change in equilibrium. . . . The majority of these patients could not swing with comfort in childhood and habitually suffered from train sickness. When rotated on a chair the larger number were uncomfortable and suffed from nausea and sweating."

The American viewpoint is in entire accord with the findings and conclusions of Dr. Head, in that the English tests, just quoted by him, are not adequate; our Air Service has used in addition—from 1917—the turning and caloric tests to determine the function of the vestibular apparatus.

3. No one denies the wisdom of making every effort to choose normal men for flying; but what does constitute the normal? Visual, auditory and general physical standards were commonly recognized and established, but vestibular standards were not. The vestibular requirements set by our Air Service were that the average normal should show between 16 and 36 seconds of after-turning nystagmus.

At the time of the adoption of these standards it was recognized as a very difficult matter to state dogmatically what is the average length of nystagmus and past-pointing. All that could be relied upon were deductions from clinical experiments with a series of healthy individuals examined by various observers over a period covering ten years, as contrasted with the impaired responses observed in merely a few hundred pathologic cases. It was realized that it was a great responsibility to establish what should be regarded as normal responses. We therefore publish at this point the composite results of the turning chair test performed by skilled standardized otologists in the examining units on many tens of thousands of applicants for the aviation service. A compilation of statistics has been made, the digest of which, with respect to responses in nystagmus, past-pointing and falling, confirms the standards originally set.

For detailed statistics the reader is referred to the book, "Air Service Medical," published by the Division of Military Aeronautics, pages 83 to 94, inclusive. The average duration of nystgamus of the entire number examined and tabulated was, after turning to the right, 23.5 seconds; after turning to the left, 23.2 seconds. The average number of past-pointings was between three and four past-pointings of each arm in each direction.

It is interesting to note that these figures are quite at variance with the findings of Barany, who stated that the average duration of nystagmus after turning to the right is 39 seconds and after turning to the left, 41 seconds. The first one to call attention to the inaccuracy of these findings was George McKenzie, of Philadelphia, who in 1917 (Observations on After Turning Nystagmus, by George W. Mackenzie, Philadelphia. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, June, 1917) corrected Barany's work and stated that 24 seconds is the normal response.

Of those rejected, 50 per cent failed in the eye requirements, as compared with only 6 per cent in the vestibular tests; and yet Dr. Henry Head states, "American workers have laid great stress on this response to rotation . . . in fact, this method of examination formed, at one time, the determining test for admission to their Flying Service." This

statement, of course, is simply due to unfamiliarity with the official examination blank (A. G. O., 609). This statement also infers that our point of view has altered; our standards are unchanged.

4. It is an idle task to speculate on the relative importance of each of the special senses to the aviator. They are all important. The need of a normal vestibular mechanism in motion sensing, however, was illustrated by experiments conducted with deafmutes on the flying field of Mineola, Long Island. These have demonstrated that those deafmutes who lack vestibular function when taken up in a plane blindfolded, could not tell the character and nature of simple evolutions. Furthermore, when taken up repeatedly it was impossible for them to develop any proficiency in motion sensing. These were checked by identical maneuvers with normals. These normals were able to detect the maneuvers much better than the deafmutes on their first trial, and further, with a few repetitions they quickly acquired more and more ability to detect these evolutions.

The one big asset to the flier is confidence. Until he has educated his vestibular sense he cannot have this confidence. Edward Rickenbacker states:

"I remember when I thought it was time to try a vrille or tail spin. I knew what I was supposed to do. I knew you put the stick over and crossed the controls, but I'd never seen anybody do it. I went up about 12,000 feet, got off some distance from the field, and flew around there for every bit of thirty minutes trying to get up my nerve to try the trick, but too scared to begin. At last I said to myself, 'What's the matter with you? You've got to do this,' and threw the stick. She went into the spin all right but I had her back to neutral after just one whirl, and I tell you I was glad when she righted. Next day I went out, and it took twenty minutes to make up my mind again. It was only on the third day that I went at the job with any confidence and let her do a real spin.

"That is still more or less the way I feel about doing a new stunt, after all the flying I have done at the front since. When you try a new thing, you are never quite certain how the machine will behave, and though you may have confidence in your ability to get out of anything as long as you have

the altitude, there is a sort of hesitation, both in the machine and in you. The trouble is, you do not know what the strains will be on either."

Every aviator has gone through exactly the same experience; but little by little he becomes familiar with these queer sensations until, fully trained, right in the midst of a spinning nose dive or tight spiral, instead of wondering "Will I ever come out of this?" he is thinking, "I wonder where I mislaid those keys this morning?"—or some such trivial thought. Acrobatics becomes a matter of pleasure rather than of fear.

The orientator is a new additional safeguard in aviation; it also provides a new method of vestibular studies. The inventor of this apparatus, William Guy Ruggles, has perfected a "turning chair" that will revolve in all conceivable planes about a central point. From a purely theoretic standpoint, he had the vision to conceive of an instrument that would permit an individual to do the "otologic part" of flying before going into the air. Furthermore, this instrument is not merely otologic—it also trains the eye and all sonses concerned in motion sensing. This machine does not teach the pupil to fly, but does duplicate all the stimulations produced in spins, spirals, loops and all turning evolutions possible in the air. A very useful feature is that the orientator produces much more violent and unusual turning sensations than can possibly be encountered in the airplane. In this way the pupil is safeguarded, by becoming familiar with sensations more intense than he can possibly encounter later in the air.

Many deaths and crashes in the future will be avoided by "orientating" the individual on the ground rather than by "throwing him into the air" to learn it.

From the purely otologic standpoint, this instrument is a great step in advance in the study of the physiology of the vestibular apparatus. From the ear standpoint, aviation has taught us what we could never have learned on the ground, by putting the human being in a fluid medium and subjecting him to twistings and turnings otherwise impossible. Medical experience in aviation has taught us that otologically the ideal flier is one whose vestibular mechanism is sufficiently acute to detect motion keenly; even more important, that this mechanism shall not be hypersensitive, rendering it difficult

for him to be comfortable and safe after turning evolutions; in other words, one who is within reasonable limits of the normal. Taking such an individual, training him to become familiar with turning sensations by means of the orientator, and then permitting him to undertake aerial acrobatics—this is the safe and sane method to begin "learning to fly."

What is the next step for future research? Fliers should be trained blindfolded, both in the orientator and in the airplane. Sufficient study has already been made to indicate that in this way the flier becomes more and more keen in the sensing of motion as well as comfortable and efficient while flying at night or in a cloud. Robert J. Hunter (Transactions of the Section of Otology of the College of Physicians of Philadelphia, May 21, 1919) conducted such experiments as Flight Surgeon in the Air Service.

Quoting from Hunter, "Blind men were used in London, during the war, to help locate the Gothas. They could hear them quickly and detect the direction better than men with sight. Silk workers in India detect the slightest variation in thickness with great skill, through the development of their tactile sense. Tea and brandy tasters for the trade have the sense of taste trained. Many here tonight recall the blind man who for years delivered provisions in the downtown sections of this city. I remember well his queer call, and especially the wonderful accuracy with which he found his way through the street. His sense of direction was highly trained."

A new pilot will always dodge a cloud. In the days when protection was of such value in war flying, airplanes could easily hide in thick ground mist, clouds or "smoky horizons"; but it was an unpleasant experience for new men to try it. New to the sensation of having their feet taken from the ground and flying in the air, depending on their "motion sensing," which had never been called upon to take up duties of this character, pilots, in self defense, had to learn this "something" which when acquired meant comfortable flying. This "something" seems to the practical flier to be educating his motion sensing. The eye, deep sense and touch had to be educated in this new environment, but it is to be noted that pilots in clouds become accustomed to cloud flying and instead

of avoiding clouds actually make for them; this indicates that some sort of adaptation has been made possible which makes the pilot comfortable after all his vision has been taken away. Pilots who have done a lot of cloud flying state that each time they enter a cloud it becomes less difficult to keep in a flying position.

This then is the next step—to train fliers blindfolded. The instructor can watch the progress of the pupil, and it is reasonable to expect that sufficient instruction can be given in this manner so that a pilot may be assured of increasing ability to fly in clouds or in any condition in which his vision is restricted or entirely eliminated. In this way the education of the vestibular sense would not only familiarize the flier with vertigo sensations but will develop an increasing accuracy in sensing motion.

So much for aviation. Is it not remarkable—the extent to which education in motion sensing can be carried? A man sits on a chair, the hind legs of which are placed upon a rope stretched across a space of thirty feet; he can sense each infinitesimally small motion so accurately that he is able to maintain equilibrium for an indefinite period. However, no matter how constantly a man might practice, he would always be laboring under a marked handicap if he does not possess all fundamental requisites for motion sensing. Conversely, with full sensory equipment, it is astonishing how quickly an individual may become adept in the instantanous correct motion sensing so essential to any motor coordination requiring speed, delicacy and accuracy.

The education of the blind is accomplished by hypereducation of his other senses—and he is then able to live comfortably in spite of his blindness. The education of the deafmute consists in hypereducation of the other special senses. These individuals are able to compensate for their misfortune by the hypereducation of the other senses which are normal. On the other hand, an individual possessed of full motion sensing acuity of vestibular sense, touch, deep sense and eye sense, can be hypereducated. The ability to translate motion sensing into motor coordination can be hypereducated to a degree that will distinguish this individual from others who are also normal. This normal is then enabled to become—by intensive

hypereducation—a tight rope walker, a trapeze equilibrist, a hotel waiter balancing a tray, a stunt flier, an expert in the orientator, a whirling dancer, an acrobatic tumbler, an expert swimmer or an old "sea dog."

Perhaps the most common example of the education of the vestibular sense, with which all people are more or less familiar, is seen in overcoming seasickness. Seasickness is now recognized to be a phenomenon resulting from movement of the endolymph within the internal ear. All forms of motion —rotary, linear fore and aft, and linear up and down—are encountered on a ship in a storm at sea. Unfortunately to get used to the sea, we must go to sea. Every effort has been made by innumerable investigators to make life at sea more comfortable for a "land lubber," only to find that, after all, the only cure for seasickness is the education of the vestibular sense.

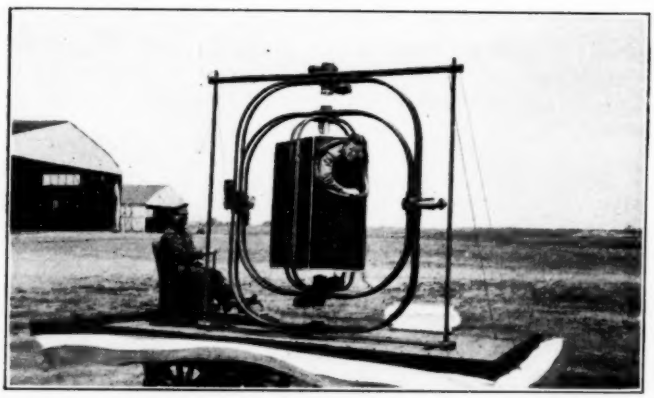

Figure 1.

Figure 2.

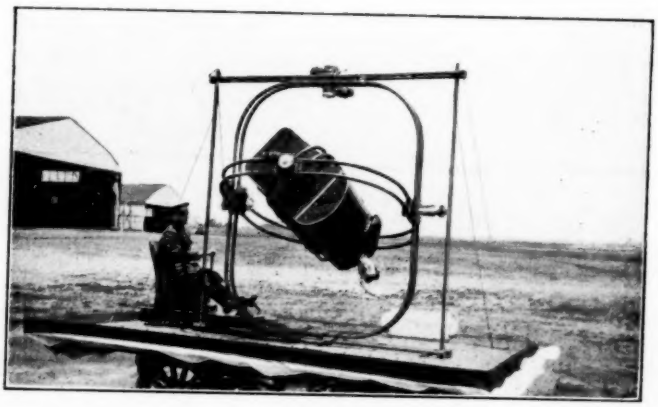

Figure 3.

LXXVIX.

NEUROOTOLOGY.

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Neurootology is a study of the internal ear and its associated intracranial nerve pathways. While it is strictly an ear study and essential to the proper diagnosis of any pathologic ear condition, it is of much broader application as a means of obtaining data for diagnosis and treatment in general medicine and surgery. The internal ear consists of two distinct organs, the cochlea, which is the organ of hearing, and the kinetic static labyrinth, which is the organ of equilibration. The cochlear portion offers a very limited field from a diagnostic standpoint, while the examination and study of the kinetic static labyrinth, consisting of the utricle, saccule and the three semicircular canals, together with their intracranial nerve pathways, affords, on the other hand, a very wide field for diagnosis and treatment.

We are indebted to the Vienna group of otologists for the present methods of examination of the internal ear. Robert Barany was awarded the Nobel prize in 1915 for his original work in this field. His name will be forever associated with the clinical relation of the internal ear and the central nervous system.

For centuries we have recognized only five special senses. Within the last two decades, a sixth sense, the muscle, joint and splanchnic sense, has been recognized. It is unlike the other special senses in that it does not make its impression on our consciousness. By means of this sixth sense the individual performs coordinate acts automatically and unconsciously. The researches which have been made have revealed a seventh special sense, the kinetic static sense, which is a special sense, just as truly as sight and hearing are special senses.

The seventh special sense, in common with the other special

senses, is presided over by a special sense organ, which likewise has an end organ, a brain center and nerve pathways connecting them. The end organ is in the labyrinth, and the brain center has been postulated by Mills to be located in the posterior portion of the first and second convolutions of the temporosphenoidal lobe near the cortical center for hearing. It is believed to be more highly developed on the right side.

The nerve pathways or vestibular tracts have not been entirely worked out. From anatomic and physiologic studies and from operative and postmortem findings these tracts may be said to have a definite course from the labyrinth to the cortical center. The fibers from the horizontal canals and those from the vertical canals join the fibers from the cochlea in the vestibule and form Scarpa's ganglion, which gives origin to the eighth nerve. The eighth nerve passes through the internal auditory canal to the brain stem, where it divides into the cochlear portion and the vestibular portion.

The fibers from the horizontal canal and those from the vertical canals have a different course within the brain stem. The horizontal canal fibers go to Deiter's nucleus in the upper part of the medulla and here divide into two separate tracts. The vestibulocerebellocerebral tract ascends by way of the juxta restiform body in the inferior cerebellar peduncle to the dentate nucleus in the cerebellum. The vestibuloocular tract ascends to the posterior longitudinal bundle and goes to the sixth and third eye muscle nuclei in the pons. The fibers from the vertical canals ascend into the pons and divide into two tracts. The vestibulocerebellocerebral tract goes to the dentate nucleus in the cerebellum by way of the middle cerebellar peduncle, and the vestibuloocular tract ascends to the fourth and third eye muscle nuclei in the pons.

The vestibulocerebellocerebral tracts ascend from the dentate nucleus through the superior cerebellar peduncle and the crura cerebri to the pyramidal tract and hence to the cortical center of the opposite side. A few of the fibers go to the cortical center of the same side. The cortical center for the quick component of nystagmus has not been definitely located, but the cerebrooocular or supranuclear tracts descend through the internal capsule to the posterior longitudinal bundle and

hence to the eye muscle nuclei. The vestibuloocular and the cerebroocular tracts preside over nystagmus, and the vestibulocerebellocerebral tracts preside over vertigo.

When the endolymph in the semicircular canals is stimulated and made to move, two reactions occur in a normal individual, namely, nystagmus and vertigo. Past pointing and falling are the objective evidence of the subjective vertigo. The stimulus is brought about by turning the individual or by douching the ears with cold or hot water for the purpose of changing the temperature of the endolymph. The turning and the temperature changes produce a movement of the endolymph in a given direction. The turning is applied by placing the individual in a specially adapted chair and turning with the head in the proper position, at a certain rate of speed and for a definite number of times. In a normal individual nystagmus and vertigo of a proper type and duration will invariably result. If one or both are absent or changed in character it has pathologic significance. This means that the stimulation did not go through so as to produce a normal reaction.

The other method of applying stimulation to the endolymph is by douching with cold water at 68 degrees or hot water at 112 degrees. This procedure results in the same phenomena that we obtain from turning, namely, nystagmus and vertigo of a given type and duration. Here again if the reaction departs from the normal or is absent it has pathologic significance. Turning stimulates the endolymph in both labyrinths, while douching stimulates only the labyrinth douched. The reaction from the cold douch is the opposite from that obtained from the hot douche.

It is important to remember that a neurootologic examination not only investigates the kinetic static labyrinth, but also its widely distributed nerve pathways. We can from such an examination gain information regarding the pathology of the labyrinth, eighth nerve, medulla, pons, the six peduncles, the cerebellum, the crura cerebri, the pyramidal tracts, the internal capsule and some parts of the cerebrum. It is this wide distribution, all so wonderfully coordinated in health, that has opened up new possibilities to the clinician. There is, in fact, no part of the body musculature unaffected by the ear stimulation. When the reaction is normal it means that the nerve

pathways are intact, but when there is a changed reaction or an absence of reaction it means an impairment of either the terminals or the lines of communication.

To the clinician the study of vertigo is an ever present problem. We are dependent for a perfect balance mechanism on the sight sense, the muscle sense and the kinetic static sense. This sense triology makes possible estimations of all sorts, orientation, locomotion, the rate of motion, estimation of weight and the realization of posture and position. The kinetic static sense is most important and it has as its sole function the maintenance of balance. After impairment or loss of one of the three, compensation may occur to a certain degree. The tabetic may learn to avail himself of his sense of sight and his kinetic static sense. The blind man is able to walk until deprived of his muscle sense or his kinetic static sense. Deafmutes in whom the kinetic static sense is destroyed maintain their balance by sight and muscle sense, and develop incoordination only in the dark or in the water. Full compensation, however, cannot develop unless two of the triology remain normal, and perversion of any one of them may be more provocative of trouble than its loss.

Previous to the realization of this important new function of the ear we regarded the ear as purely an organ of hearing. The physician was concerned with the disturbance or loss of the hearing function or with the effects of the extension of infection from the ear to adjacent structures. The general practitioner is concerned with the new development in diagnosis because of the value of the ear tests in determining the cause of vertigo, no matter what its origin or type, and in recognizing pathologic conditions of the internal ear itself. Vertigo is a subjective sensation of disturbed relationship of one's own body to the surrounding objects in space. Although it has been recognized for many years that vertigo may result from ear disturbances, the conception that all vertigo, from whatever cause, is peculiarly an ear study, is the result of the study of the new otology. These dizzy cases suffer acutely with apprehension and, with no explanation at hand, their apprehension increases. The vague terms in which vertigo is often spoken of is an index of the uncertainty in our minds regarding this distressing condition. Often we speak of intes-

tinal or stomach vertigo or dizziness from refractive errors or from Bright's disease or neurasthenia without thinking of the real mechanism of its production.

It has been impossible to tell, in any given case, whether the vertigo was due to a functional or organic disease and still less to recognize whether it was of trivial significance or the forerunner of some serious disease. Vertigo should be considered as a distinct clinical entity, deserving as careful investigation and analysis as any other distressing pathologic condition.

All conscious sensations are cerebral. Vertigo is a disturbance perceived within the brain, just as taste or smell. If a disturbance of any organ is accompanied by vertigo it is because of a direct attack on the apparatus capable of producing vertigo. If the same condition fails to involve the ear and its pathways, there will be no vertigo. If a patient with vertigo gives an exaggerated reaction of normal character, it means that the patient is suffering from an irritation of the vestibular organ, either distally or centrally. This is usually toxic in character. The source of the toxicity may be a focal infection or a gastrointestinal toxicity or some specific disease, such as syphilis or any of the acute infections. If the patient gives an abnormal reaction it signifies an organic lesion. It is assumed that any toxicity may in time produce an organic change in the tissue involved. It has been proven beyond doubt that vertigo is not a "cerebellar pull," but the cerebrum's conscious effort to compensate.

The following classification of vertigo is, as far as we know, complete:

1. Involvement of the ear mechanism by a lesion in the ear itself. "Meniere's symptom complex" is due to a sudden destruction of the labyrinth by hemorrhage or by effusion into it, such as may occur in nephritis or diabetes or other conditions affecting the cardiovascular system. The usual symptoms of deafness, tinnitus, vertigo, staggering and, if severe, nausea and vomiting, are all explained by either a toxic irritation of the labyrinth or by cariodvascular disease producing anemia or hyperemia. Any inflammatory condition affecting the labyrinth gives the same picture in a varying degree. Slow

degenerative changes within the labyrinth cause attacks of vertigo from time to time.

2. Involvement by a lesion affecting the intracranial pathways from the ear. Lesions within the brain causing vertigo may be tumor, hemorrhage, thrombosis, infarct, abscess, gumma, tubercle, leukemic infiltration, specific neuritis, multiple sclerosis, syringomyelia, polioencephalitis and meningitis.

3. Involvement by ocular disturbances, either through the eye muscle nuclei or through association fibers from the cuneus to the cortical terminus of the fibers from the ear in the posterior part of the first and second temporal convolutions. This group includes all the cardiovascular conditions which produce either congestion or ischemia within the cranium.

4. Involvement by toxemias from any organ or part of the body. This group includes ptomaine poisoning, alcoholism, chemical poisoning, such as lead, quinin, salicylates, the toxemia of nephritis, rheumatism (focal infections), syphilis, infectious fever, such as mumps, scarlatina and typhoid fever. These toxemias may be grouped in two classes: (a) Evanescent toxemias, which have caused no degeneration of the cellular elements within the ear or the nerve pathways. This constitutes a large group of the cases of vertigo seen by the practitioner. (b) Toxemias which have caused a definite damage to the internal ear or its nerve extensions, such as in mumps or syphilis, or repeated attacks of a milder toxin, such as from gastrointestinal conditions or focal infections.

In aviation we have a practical example of the function of the ear in maintaining equilibrium. It is not necessary for an individual to have a perfect muscle sense, perfect sight and a perfect kinetic static sense in order to have an equilibrium sufficiently good to take care of the ordinary pursuits of life. In aviation, however, it is imperative. When flying above the clouds or in the dark the sight sense is of no avail, and the muscle sense in an unstable and moving machine affords little or no sense of balance. It is essential, therefore, that the aviator have a perfect vestibular apparatus, as it is upon this alone that his balance when flying depends. Some of the unexplained accidents in aviation are very likely due to a sudden concussion of the internal ear from the deafening

roar of the motor or by a decrease in the air pressure at great heights. In a rapid ascent from a denser to a rarer air there occurs an oxygen insufficiency which has a direct effect on the ear mechanism through the blood stream. Unquestionably many an aviator has gone to his death because he did not know that he did not possess a normal internal ear. The United States was the first nation to appreciate the need of the neurootologic examination of aviators. During the late war this branch of the service was very highly and efficiently developed. The world owes a great deal to Isaac Jones of Philadelphia, not only to his pioneer work in this field during the war, but as well to his original researches and contribution to neurootology for the past few years.

Seasickness or mal-de-mer is a phenomenon resulting from the movement of the endolymph within the internal ear. As long as man has traveled by water, seasickness has been a source of distress to thousands of travelers. It would be impossible to enumerate the theories advanced for the cause of the malady, and the remedies have been as numerous as the theories. With the discovery of the physiology of the kinetic static labyrinth and a study of the reactions attendant upon stimulation of this organ the true etiology of seasickness was evident. The first symptom of seasickness is dizziness, which is followed by staggering on attempting to move. The face becomes flushed and soon is changed to a pallor, followed by nausea, and if the condition continues, by vomiting. This is entirely due to the endolymph movement brought about by the motion of the ship. This is the same picture obtained by douching the ears. Women are more susceptible to seasickness than men, little children are very little affected and infants not at all. This corresponds with our experience following douching. Infants never vomit, no matter how long the ears are douched. The vestibular organ of infants is not fully developed, which accounts for the absence of this reaction. From repeated experiences most people develop what may be termed an immunity to seasickness, which is also true of repeated stimulation by artificial means. An individual who cannot be nauseated by the examination of the vestibular apparatus will not become seasick.

The ordinary endolymph movements, such as accompany the usual pursuits of life, are responsible for our body balance because the brain through countless repetitions has learned to interpret the significance of these movements. When the movements become unusual the brain cannot interpret them and confusion and vertigo result. If the unusual movements recur frequently the brain learns to interpert them and vertigo no longer occurs, hence immunity to seasickness. The mechanism of vomiting is chiefly through the vagus and phrenic nerves. Nausea is a different reaction and is the cerebral interpretation of the unusual stimulus. Cumulative nausea may produce vomiting by a motor impulse from the cerebral cortex to the nuclei of the vagus and phrenic nerves. Besides the cerebral impulse we have association fibers directly from the ear to the vagus and phrenic. The vestibular nerve enters the posterior longitudinal bundle, which in turn is directly connected with the vagus nucleus. Other vestibular fibers enter the medulla and descend to the motor cells of the anterior horns of the spinal cord. The phrenic nerve has its origin in the cells of the anterior horns of the second, third and fourth cervical vertebræ. It is thus that the impulse travels direct to the vagus. Certain olfactory and visual impulses are contributing factors in overcoming the cerebrum's attempt to keep from vomiting. We may, of course, have nausea and vomiting without endolymph movement. If one could place himself in such a position that the motion of the ship would cause endolymph movement only in the horizontal plane there would be no seasickness. Unfortunately this is impossible, as the ship movements are in more than one plane. However, the seasickness is often mitigated by lying in the plane of the greatest movement. Any treatment or drug that will lessen nerve irritability will tend to lessen the seasickness. By a vestibular examination we can determine if an individual will become seasick.

It is well known that the toxin of syphilis has a predilection for the eighth nerve, and oftentimes this will be a very early manifestation of the disease. In any stage of the disease it will often be the first part of the nervous system to become involved, and this may occur in the entire absence of any other clinical evidence of the disease. In the past we have

had to depend upon the functional tests for hearing to detect the presence or absence of eighth nerve involvement. At the best this is uncertain, and its unreliability has placed this method of diagnosis in ill repute. In the examination of the labyrinth by the Barany method we have an objective measurement of the degree of function of the eighth nerve. The following data may be presented as an aid in the diagnosis and treatment of syphilis. In the absence of the microscopic diagnosis of the initial lesion, or a Wassermann reaction, we can by these tests detect a slight diminution of function of the eighth nerve and by repeated examinations detect a progressive condition. In this way we may gain information that is suggestive in the presence of an uncertain Wassermann. We may detect by this means a lesion of the eighth nerve or the central nervous system which might not be evident for some time. In some cases this has been demonstrated years before any clinical evidence, and in the presence of a negative spinal fluid Wassermann. The early diagnosis of syphilis of the central nerve system is so important that the use of this means of diagnosis cannot be too strongly urged. We may demonstrate this before gross changes have taken place and while there is still hope of therapeutic help.

Recurrence of syphilis of the central nervous system is very insidious. The blood and spinal fluid Wassermann may be negative. The inconvenience of repeated spinal fluid examinations for control of recurrences is such that it is often not done, but this method affords an opportunity for repeated tests without discomfort or inconvenience to the patient. We may also by repeated examinations during the course of treatment of syphilis of the nervous system determine the efficiency of the treatment. We know how difficult this measure is by the usual means. If the patient is not cured the vestibular examination of the central nervous system will accurately reveal it.

The value of the eye examination to the neurologist has long been recognized. Based on opinions of neurologists and ophthalmologists who have had experience with the Barany examination, it is safe to say that the ear examination affords as a rule more definite and exact information than the eye. In Vienna no neurologic examination is considered complete

without a vestibular examination. The ear tests are very valuable in making differential diagnoses between labyrinth and intracranial lesions and in furnishing additional data for intracranial localization. Nystagmus and vertigo, often associated with nausea and vomiting, may be due to either a labyrinth lesion or an intracranial lesion. In many instances the symptoms of internal ear disturbance and cerebellar lesion are identical. It is in such cases that the ear tests are valuable.

A differential diagnosis between peripheral and central lesions by means of the ear tests depends on certain general principles. A peripheral lesion of the labyrinth or eighth nerve is suggested by the following:

1. An impairment of the function of both the cochlear and the kinetic static labyrinth. If the examination of the hearing shows cochlear deafness, and the tests of the semicircular canals show that their function is also impaired, it immediately becomes probable that we are dealing with an end organ lesion.

2. The history or presence of tinnitus is evidence of labyrinth involvement, but its absence does not mean an unimpaired labyrinth.

3. Proportionate impairment of the responses from the horizontal canals and of the vertical canals indicates labyrinth involvement.

4. Proportionate impairment of both nystagmus and vertigo from the horizontal canals and normal responses from the vertical canals indicates a lesion involving the fibers from the horizontal canal in the eighth nerve or in the horizontal canal itself. It is the proportionate impairment of responses that speak of an end organ lesion.

A central lesion is suggested by the following:

1. Normal cochlear, but impaired or nonresponsive semicircular canals.

2. Normal responses from the horizontal canals, but absent responses from the vertical canals.

3. Normal responses from the vertical canals, but absent responses from the horizontal canals.

4. Normal vertigo, but impaired nystagmus from the horizontal canals.

5. Normal nystagmus, but impaired vertigo from the horizontal canals.

6. Normal vertigo, but impaired nystagmus from the vertical canals.

7. Normal nystagmus, but impaired vertigo from the vertical canals.

8. Normal vertigo and normal nystagmus from any semicircular canal, but impaired past pointing in any direction of any extremity.

9. Normal vertigo and normal nystagmus from any semicircular canal, but an impairment or absence of normal falling.

10. Spontaneous vertical nystagmus is pathognomonic of a central lesion and is indicative of involvement of the brain stem caused either by infiltration or pressure. A lesion of the labyrinth may produce many forms of spontaneous nystagmus, horizontal, rotatory, oblique or mixed nystagmus of these types, but an ear lesion can never produce a spontaneous vertical nystagmus, either downward or upward.

11. If there exists a spontaneous nystagmus to the right and nonresponsive semicircular canals of the right ear, an intracranial lesion is suggested. The nonresponsive labyrinth, if the labyrinth alone were responsible, would produce a nystagmus to the left.

12. A spontaneous nystagmus of increasing intensity or of long duration is indicative of a central lesion. A spontaneous nystagmus due to a lesion of the labyrinth shows its greatest intensity at the onset of the disease, becoming less and less marked and disappearing after a few days.

13. If the stimulation of any semicircular canal produces a perverted or inverse nystagmus, it is pathognomonic of a central lesion and is indicative of a brain stem involvement. Such phenomena as the following are frequently seen: Douching the right ear with cold water, with the head back, thus stimulating the right horizontal canal, should give a pure horizontal nystagmus to the left. If on such stimulation there occurs a vertical nystagmus upward or downward, a rotatory, oblique or mixed nystagmus, it may be spoken of as a perverted nystagmus. If instead of a horizontal nystagmus to

the left there is produced a pure horizontal nystagmus to the right it may be termed an inverse nystagmus. Neither a perverted nor an inverse nystagmus can possibly be produced by a lesion of the labyrinth or eighth nerve. A peripheral lesion produces a poor nystagmus or no nystagmus, but an absolutely false response of necessity demonstrates a central lesion.

14. If the ear stimulation produces a conjugate deviation of the eyes instead of nystagmus, it is pathognomonic of a central lesion.

The above outline indicates how additional data may be furnished to the neurologist by the labyrinth examination in determining whether he is dealing with a lesion of the internal ear, the eighth nerve, the brain stem or the cerebellum. In the broad field of brain localization the examination of the internal ear is also of distinct value. The particular feature of the ear examination is that the aurist sends in a stimulus to the brain centers and then notices the responses of the different parts of the body to this stimulation. If the ear and its nerve pathways are all intact the responses will be normal. If there is a failure of any or all of the responses, it is positive evidence of an interruption along that particular path or paths, that fails to bring about these responses.

In order to utilize the knowledge obtained from these tests it is essential to bear in mind the various pathways constituting the vestibular apparatus. The horizontal canal fibers after entering the brain stem have a different course from the fibers from the vertical canals. Each tract divides into two tracts, one the vestibuloocular tract, responsible for nystagmus, and the other the vestibulocerebellocerebral tract for vertigo. By determining the specific tract or tracts which fail to respond we can locate the site of the lesion. In order for the data to be reliable it is essential that the technic of the examination be accurately and painstakingly carried out. The ear examination is not for the purpose of making a neurologic diagnosis, but merely to give additional information, by a series of refined experiments, to be added to the knowledge obtained by the neurologist by other methods at his command. In the cases which the neurologist finds no difficulty

in making a diagnosis it is useful in providing an additional evidence in corroboration. In the obscure case the neurologist may find the ear tests to provide the most accurate information obtainable.

The diagnosis of the precise location of a lesion within the cranium is probably the most difficult task with which the surgeon is confronted. Realizing this, he avails himself of every modern aid and diagnosis, such as the laboratory and spinal and corpus callosum punctures and the refinements developing therefrom, such as oxygen injections, pressure measurement and the analysis of the fluid withdrawn and the X-ray. For many years past no surgeon would consider doing an intracranial operation without an eye examination. We have previously shown the value of the labyrinth examination in differentiating between labyrinth and intracranial lesions. A point worthy of emphasis is that with the aid of these tests he is frequently enabled to determine whether a lesion is operable or not. Many lesions of the medulla, pons or cerebellar peduncles, which are inoperable by the very nature of their location, will show pronounced cerebellar symptoms, and not infrequently the cerebellum is exposed in such cases in the hope of finding a lesion near the cortex and removing it. For such a differentiation, in many cases, no method so far developed equals the Barany tests. If after turning and douching there appears a normal past pointing of both extremities in both directions, it may safely be assumed that the cerebellum is intact. This cannot be regarded as absolutely final, but is much more definite than any other known method for determining the integrity of the cerebellum.

Tumors of the cerebellopontine angle either originate from one of the cranial nerves in the angle, usually the eighth nerve, or have invaded the angle secondarily from the cerebellum or brain stem. The eighth nerve is usually involved in these cases, and our study of the eighth nerve gives a direct insight as to the condition in the cerebellopontine angle. In addition, the ear tests usually demonstrate the two following phenomena in cases of cerebellopontine angle tumor:

1. An absence of all responses from the vertical canals of the ear opposite the lesion. Given a tumor of the right cerebellopontine angle, the usual findings are as follows: The

right ear gives no responses, the cochlear showing an absence of all function and the stimulation of the horizontal canal and the vertical canals failing to produce any nystagmus, or vertigo, or past pointing or falling. The left ear shows unimpaired hearing and the horizontal canal normal nystagmus, vertigo, past pointing and falling. The vertical canals, however, fail to produce any response because the vertical canal fibers are most probably impaired owing to pressure upon the pons by the tumor of the angle.

2. Crossed past pointing. This phenomenon consists of persistent past pointing of both upper extremities either outward or inward, regardless of the ear stimulation employed.

In the examination of head injury cases we are able to be of considerable assistance. If the injury involves the labyrinth, as is so often the case, we can determnie accurately the extent of the damage and give an accurate prognosis. In another group of head injury cases in which the intracranial hemorrhage is the main factor in producing a serious intracranial condition, we have found a very consistent picture in our neurootologic examination. In this group the blood, regardless of the seat of the injury, gravitates to the posterior fossa above the tentorium and, filling the fossa, overflows through the incisura tentorii, into the fourth ventricle. The examination of a very considerable group of such cases has given consistent vestibular findings. Stimulation of the vertical canals gives no responses whatever. We have an absence of vertigo and nystagmus showings that the stimulation does not go through. Stimulation of the horizontal canals gives either a normal reaction or, if the hemorrhage is large, a diminished vertigo, but a normal nystagmus. In other words we have a central blocking of the passage of the stimulus. The interpretation of the above findings locates the obstruction in the fourth ventricle. The vertigo which we have in these cases is explained by the vestibular examination. The prognosis is good in most cases, as compensation occurs and the patient does not have vertigo under ordinary circumstances. The vestibular examination may be made use of in helping to determine the advisability of surgical intervention in these cases. If the vestibular findings are normal it means that there is not sufficient hemorrhage to cause the condition

above described. In the absence of blood in the spinal fluid and no increase in the pressure, a normal vestibular examination adds weight to the surgeon's decision of nonsurgical treatment.

If the ear stimulation produces normal nystagmus, vertigo, past pointing and falling, the Barany tests are of unquestioned value in eliminating a lesion in the posterior fossa and brain stem. If they render no other service than this it would be sufficient to make them a distinct contribution in the diagnosis of intracranial lesions. In addition they may prove helpful in preventing unnecessary operation. In fact, no operation on the brain should be undertaken without giving the patient the benefit of the ear examination.

The intimate relation between the ear and the eye can best be appreciated when we realize that the ocular mechanism constantly depends upon stimuli from the ear for precision of movement. Steadiness of fixation is dependent to a large extent on normally functionating ears. Tonic impulses from the right ear, for example, continually tend to draw the eye to the left. This is proven by a sudden loss of function of the right internal ear, which invariably results in a deviation of the eyes to the right because the tonic impulses tending to draw the eyes to the left are impaired and there is a resulting nystagmus to the left. The same is true of the left side, only in an opposite direction. This may be further demonstrated experimentally by electricity. By applying the anode of the galvanic current to the right ear, which depresses the function, there results a drawing of both eyes to the right, with resulting nystagmus to the left. By applying the kathode, thus stimulating the right ear, the opposite phenomenon results. It is thus shown that ocular equilibrium, just as bodily equilibrium, is normally dependent on properly functionating ears. Artificial stimulation of the ears will produce to order any type of nystagmus. The laws governing these phenomena are invariable and simple. For example, if we wish to produce a vertical nystagmus upwards, we place the patient's head over towards the right shoulder, turn him to the left and we have a vertical nystagmus upward. The fistula test also serves to illustrate. If there is a caries of the bone

in the outer wall of the labyrinth or a movable stapes, applying pressure or suction in the ear canal causes nystagmus. The tests have a practical value to the ophthalmologist in studying ocular palsies and spontaneous nystagmus.

In studying ocular palsies it is important to note that the ear stimulation is much stronger than the voluntary cerebral stimulus. If a patient is unable to look to the right with the right eye, the question arises as to the origin of the involvement of the sixth nerve. If ear stimulation causes the eye to move outward it is evident that the sixth nerve is not completely destroyed. This makes for a refinement of the determination of the degree of paresis and aids in the prognosis. The horizontal canal ocular fibers go to the sixth nerve, which supplies the external rectus and that part of the third nerve which supplies the internal rectus. If appropriate stimulation is applied it will show, in many cases, that the patient's inability to look to the right or left is due to only a partial impairment of the sixth nerve. The same will apply to tests for paralysis to the right or left or upward or downward. Loss of conjugate deviation in various directions should always be studied by means of the ear tests. If there is a loss of voluntary control and the patient when asked is unable to look in a given direction with either eye, and the ear stimulation causes the eyes to move in this direction, it shows without question that there are normal pathways from the ear to the eye muscle nuclei, and through the nerves of the eye muscles themselves, and that the lesion is supranuclear.

The Barany tests have taught us much that we did not know about spontaneous nystagmus. In a given case of spontaneous nystagmus, the first problem is to determine its type. Is it ocular or vestibular? If the oscillations of the eyeball consist simply of a to and fro roll, similar to the swinging of a pendulum—that is, the movement or roll in one direction is just as long and just as rapid as in the other, then it is of ocular type and suggests some local eye disturbance. It may be due to any condition that prevents the rays of light from pursuing an uninterrupted course through the various ocular media, such as corneal scars or anterior polar cataract. It is the effort to look around the obstruction. Ocular nystagmus may also result from interference with the proper action

of the rays of light at the macula, provided the lesion occurred before central fixation was established.

In sharp contrast to the foregoing, if the movement of the eyes exhibit a definite rhythm, slow in one direction followed by a quick movement or recoil in the opposite direction, the nystagmus is of the vestibular type and due to an abnormal mechanism and not a lesion in the eyes themselves. It can be produced by a lesion affecting any of the pathways between the ear and the eye muscle nuclei, either within the ear or within the cranium. Such a nystagmus is not due to an attempt at fixation but to a disturbance within the nuclei themselves or through impulses from the ear to the eye which cause the eye to deviate to one side. When the eyes are thus drawn to one side, impulses from the cerebrum to the eye muscle nuclei quickly pull the eyes back in the opposite direction. The slow component is due to irritation, impairment or destruction of the pathways from the ear to the eye muscle nuclei, whereas the quick component results from the attempt of the cerebrum to correct the altered position of the eyes.

The cause of ocular nystagmus is readily determined by an eye examination, but it is those cases in which the lesion lies not so near the surface that the ear tests are absolutely essential. No case of vestibular nystagmus can be considered to have had a proper examination without the ear tests.

The importance of the examination and study of the cochlear portion of the labyrinth is accepted by all otologists. That the kinetic static portion of the labyrinth is equally, if not more, important is not yet recognized by all otologists, or at least is not practiced by even a small percentage of them. An examination of the internal ear is certainly not complete if only a portion of it is examined. The examination and study of the kinetic static labyrinth and its pathways is just as much a part of otology as the examination of the cochlear portion. It is a misconception to regard the examination of the kinetic static labyrinth as difficult to make and hard to understand and interpret.

The entire physiology of these ear tests may be summed up as follows:

1. The eyes are always drawn in the direction of the endolymph movement.

2. The vertigo is always in a direction opposite to the endolymph movement, and past pointing and falling are always in a direction opposite to the vertigo.

By knowing these laws the otologist is master of the physiology of the subject. By making this method of examination a regular part of his practice the otologist may enlarge his field of usefulness and add great interest to his work. In the routine examination of the ear the otologist is constantly confronted with cases of deafness in which the significance of the tuning fork tests is obscure and inconclusive. By determining an involvement of the kinetic static portion of the internal ear he has at once presumptive evidence of a similar impairment of the cochlear portion. The accepted method of examination for the function of the cochlea depends on the honesty, the intelligence and cooperation of the patient, while the Barany tests of the internal ear presents objective evidence of the function of the kinetic static portion of the eighth nerve.

In the routine examination it is not necessary to make all of the tests. Merely turning the patient to the right or to the left will give sufficient information to determine the necessity for further elaboration. This can be done quickly and without any disagreeable result to the patient.

When the otologist is confronted with surgical problems of the internal ear itself, as in inflammatory conditions of the labyrinth resulting from the extension of a suppurative process in the middle ear, it is generally recognized that the turning and caloric tests, and occasionally the fistula test, are indispensable in determining the nature and degree of the involvement. The tests are the only means by which the aural surgeon can determine whether an operation on the labyrinth is necessary or whether the vestibular symptoms produced by the nonsurgical affection result from a lesion of the labyrinth itself or along the intracranial nerve pathways from the internal ear. Such a differentiation can only be made by the Barany examination and such is the otologist's problem. Vertigo is essentially an otologic study, and every otologist should be prepared to analyze such cases. In the same way he should always be prepared to give the necessary information to the internist, the surgeon and the neuroolgist as the possible loca-

tion and the differentiation from an internal ear and an intracranial lesion.

In the foregoing presentation of the subject of neurootology text books and the current literature have been freely drawn upon. The section on head injury cases is, as far as the writer knows, entirely new. A separate treatment of this phase of neurootology will be presented in the near future.

121 St. James Pace.

LXXX.

BARANY TESTS IN SUPRATENTORIAL TUMORS PROVED BY OPERATION OR NECROPSY.*

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INTRODUCTION.

A slow but sincere scientific interest, a steady increase in knowledge and an indefinite amount of discussion are embodied in neurootology in general and in the Barany tests in cases of supratentorial tumor in particular. Early the neurologist looked on neurootologic localizations of intracranial lesions with interest but also with reservation. These limitations are being eliminated and will be steadily eliminated as our knowledge from proved cases multiplies. The localization by Barany tests of supratentorial tumors is one of the later developments of neurootology.

A Barany test is indicated in all cases of vertigo, but is especially useful in all lesions of the central nervous system. In performing this test it is essential that the turning and the caloric tests are not made consecutively, because the responses are modified after repeated stimulations of the semicircular canals. It is also useful for productive work that the intracranial localization of a lesion by this test is made independently and without knowledge of the neurologic localization. A report of nine cases in which the test was made follows:

Case A236828. Miss S. L., aged 40, was examined July 2, 1918, chiefly because of dull frontal headaches and loss of strength. The patient's family and personal history are neg-

*Abstract of thesis submitted to the faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Otology, Rhinology and Laryngology, May, 1920.

ative. The patient had always been well until one morning in October, 1917, while combing her hair she felt a sudden jerk in the left arm and it immediately became numb and powerless. Severe and almost continuous frontal headaches began at this time. Projectile vomiting often followed the headaches and somewhat relieved them; the left hand improved slightly. Partial deafness, which had existed since childhood, increased. The patient's limbs jerked when she was lying down.

Physical examination: The weight, blood pressure, urine and blood counts were normal. The eyes showed double choked discs of three diopters. The blood Wassermann test was negative. The spinal fluid Wassermann tests with 0.4 and 0.55 cc. were negative. The Nonne reaction was positive.

Neurologic examination, July 4, 1918: The neurologic examination was rendered difficult by the somewhat clouded mental condition of the patient. A nystagmus was noted. The cranial nerves were otherwise practically normal, with the exception of bilateral choked discs of about three diopters. Movements of the left arm were awkward as compared with those of the right. The reflexes were normal throughout, save for a slight diminution of the left abdominal reflexes. With the exception of a moderate loss of joint sensation of the left toes and an almost complete loss of joint sensibility in the left fingers, with corresponding asterognosis, sensation was normal throughout. No other significant changes were noted. A neurologic diagnosis of a tumor of the brain, located probably in the postcentral gyrus of the right side, was made.

Barany test: A Barany test revealed a moderate combined type of deafness, a bilateral spontaneous horizontal nystagmus and a spontaneous pointing fault with the left arm. The turning test showed prompt reactions throughout. The caloric test revealed, on the right, a marked and definite exaggeration of all responses from all the canals. Nystagmus began almost at once: with the test of the left verticles it was of a perverted type, with vomiting and marked vertigo which was early and severe on the right also. A pointing fault was exhibited by the left arm. Falling was to the left. The horizontal canal showed marked reactions throughout. The reactions from the left canals, which showed interference with the posterior

longitudinal bundles and the middle peduncle on the same side were interpreted as pressure signs. The diagnosis from the Barany test (Chart 1) was a supratentorial lesion on the right side, causing pressure downward.

July 16, 1918, a glioma of the brain 5 by 10 cm., weighing 63 gm. (Fig. 1), was removed from the right frontomotor area.

January 16, 1920, when the patient returned for examination, she was normal mentally and had control of the left arm.

Case A235175, Mrs. R. P. O., aged 47, came to the clinic June 14, 1918, walking but in the need of considerable support. The patient's father died of an accident; her mother is 78 and in good health. Two brothers are in good health and two died of typhoid fever and scarlet fever, respectively. One sister is well. One daughter, sixteen months of age, died of sarcoma of the kidney. The patient's menstrual history was not unusual. The last period was in March, 1918; previous illnesses were also unimportant. Her chief complaint was inability to walk for the past two and one-half years. She felt physically weak and mentally tired. Eight months' rest in a sanatorium had not especially benefited her. She had intense bilateral, frontal and occipital headaches. Early in December, 1917, the patient was in coma for three days, then became markedly apathetic and entirely lost her memory for past incidents. Early in the spring she again went to a sanatorium for six weeks and was somewhat improved, but on returning to her home her condition became worse. She had involuntaries and urinary frequency with loss of control of the sphincter.

Physical examination: The patient's weight was 175 pounds, 30 pounds above normal. Her left pupil was larger than the right. She had a double choked disc of about three diopters right and one to two diopters left. There was a moderate blurring and loss of detail about both discs. Reflexes were normal. The Wassermann and Nonne tests on the spinal fluid and the Wassermann test of the blood were negative. The lymphocytes were normal. The routine urine and blood examinations and the combined phenolsulphonephthalein test were all negative. A roentgenogram of the head showed an appar-

ent thickening of the base of the sella with obliteration of the posterior clinoid process.

Neurologic examination (June 14, 1918): Neurologic examination revealed the following status: Vision was 6/6 in both eyes; there was a moderate blurring and loss of detail around the edges of both discs, suggesting a pupillitis; the visional fields were practically normal. The left pupil was slightly larger than the right, with slight impairment of reaction to light in the former, and considerable impairment in the latter; reaction to accommodation was normal. There was a nystagmus on looking toward the right. The tendon reflexes were all moderately increased; the abdominal reflexes could not be elicited; however, the patient was obese and the abdominal walls flabby. The patient walked on a rather wide base and gave the impression of being very feeble. There appeared to be some dementia; the patient asked the same questions repeatedly, raised numerous objections to the examination, and cooperated very poorly. The spinal fluid tests were negative.

The patient was sent home to be cared for by a local doctor and asked to report by letter frequently. Her condition became definitely worse; when she returned January 31, 1919, projectile vomiting was more frequent than at the time of the first examination, consciousness was decreased, and double choked discs were increased to four diopters.

Neurologic examination (January 31, 1919): There were now definite bilateral choked discs and a nystagmus. There was a marked general weakness, particularly on the right, with a corresponding increase in muscular tonicity. The tendon reflexes were all greatly increased, the abdominal reflexes were absent, and there was a strongly positive Babinski phenomenon on the right and one doubtfully positive on the left. The mental condition was decidedly clouded, attention being too poor to make a detailed examination.

A diagnosis of brain tumor, probably located in the left frontomotor area, was made.

Barany test: The Barany test presented one of the interesting phenomenon syndromes of supratentorial tumors (Chart 2). There was no cochlear fault in either ear, nor was there any spontaneous nystagmus. The patient was in the hospital

under observation. She was unable to raise her arms, due mostly to her dementia, but somewhat also to her weakness. A turning test, therefore, was impossible.

Two features were prominent in an analysis of the caloric test, first, much exaggerated vertigo and falling, and second, absence of nystagmus from the right verticals. Pointing tests were impossible. This indicates the supreme test in the ability to interpret a Barany syndrome. The marked vertigo and falling suggest a lesion above the stem, but in this case the stem itself gave an abnormal response. The explanation is clear and in accordance with facts.

The most striking effects of supratentorial tumors or, in fact, of any brain tumor, are those of pressure. This we know may cause symptoms due to injury to remote parts of the brain, for example, a lesion above the stem may, due to its pressure, cause a paralysis or paresis of the sixth cranial nerve, resulting in a paralysis or paresis of the external rectus muscle of the eye. Similarly an enlargement of the sella turcica or an absence of the posterior clinoid process is shown by the roentgenogram. These two facts have been proved definitely in a large number of cases. The pressure in this case exerted not only a rather typical exaggeration of vertigo and falling, but also the effect of the pressure from above downward, resulting in a brain stem syndrome partially involving the posterior longitudinal bundles. From the Barany, therefore, a diagnosis was made of a supratentorial tumor in the left cerebral hemisphere.

At operation, February 8, 1919, a subcortical tumor was found in the left frontal lobe, 2.5 cm. below the surface of the cortex, involving the middle and lower frontal convolutions.

Case A233010. Mr. A. St. L., aged 48, came to the clinic October 13, 1919, because of fits which began eighteen months before. The condition affected the right foot and arm and the head, occasionally the left side. Sometimes the attacks were followed by unconsciousness. The attacks were tonic in nature, started from the foot on the right side and passed up the same side, the head turning to the right. The fits lasted about ten minutes. The attacks were often preceded by an electric shock throughout the body. Formerly they occurred about every three weeks, at the time of examination once in

seven weeks. The patient had severe frontal and occipital headaches, often lasting three days, and he complained of diplopia and tinnitus. He did not bite his tongue or froth at the mouth. He was rather dull intellectually and slow to answer questions. Sixteen years before in an explosion of dynamite a board had struck him in the left occipital region, but this had not appeared to give him any trouble.

Physical examination: The patient's pupillary reflexes were sluggish. His body was erupted with marked pigmentation. He had a bilateral choked disc of two diopters in the right eye and of one diopter in the left. The routine examinations showed the blood and urine to be normal. The blood and spinal fluid Wassermann tests were negative to 0.4 cc. of fluid. The Nonne was positive; there were five small lymphocytes.

A roentgenogram of the head failed to reveal the sella, but a suspicious area of erosion was found in the occipital region. The patient's temperature was normal.

Neurologic examination (January 31, 1920): The patient was found to have normal vision, choked discs of two diopters on the left and one on the right, the right pupil slightly larger than the left, and a slight diminution in all the pupillary reflexes. Examination of the other cranial nerves was negative. Weakness was general, but there was not any evidence of local impairment in muscular power. Coordination was moderately impaired on the right side in both the arm and the leg, and very slightly impaired on the left. The gait suggested an ataxia, and the Romberg test was slightly positive. Sensation, including sterognosis, was normal. On rest and on static innervation a coarse rapid tremor appeared in the right arm. The tendon reflexes were all moderately increased but equal on both sides. The abdominal and plantar reflexes were normal. The sphincter control was normal. Spinal fluid examination gave a negative Wassermann test, a positive Nonne, and five small lymphocytes to the cubic millimeter. The patient was strikingly sluggish mentally and indifferent in his cooperation during the examination. A roentgenogram of the head showed the outlines of the sella to be very indistinct.

A diagnosis of a brain tumor, probably of the left frontal lobe, was made, and exploration advised.

Barany test: The Barany test was very interesting. The turning was not done, because the patient was quite sick. The caloric test showed that the nystagmus time from the right vertical canals was markedly delayed. Vertigo was normal. There was no falling, and the pointing was reduced. The right horizontal canal showed increased pointing. Obviously, the turning was not necessary in this case, and a diagnosis was made of a left supratentorial tumor which was producing pressure signs on the right hemisphere (Chart 3).

The patient was operated on November 27, 1919, and a large osteoplastic flap was turned on the left temporal area. Profuse bleeding was encountered, the dura near the midline bleeding freely. The ventricle was entered twice with a trocar, but only a few drops of fluid escaped. In making the stabs it felt as if there was a tumor under this area of the dura; the dura was incised and a tumor measuring 7 cm. by 5 cm. was found, very closely adherent to the dura but encapsulated on the brain side. Enucleation was difficult and bloody, a large portion of the dura, about 4 cm. in diameter, was excised attached to the tumor (Figs. 2 and 3), which extended up to the midline and made pressure on the flax cerebri. The longitudinal sinus was probably entered, as there were marked venous hemorrhages which required packing. The excavation in the brain was filled with animal membrane held in place by a silk suture. The operator noted that a cerebral hernia would develop due to the dural defect.

The patient recovered and had a resection of the small cerebral hernia. The tumor, 6 cm. in diameter, on pathologic examination, was shown to be endothelioma.

The findings in this case were definite and instructive. The exact localization of the lesion was made from the caloric test alone. The pressure signs on the left hemisphere were verified by the location of the tumor. The patient had no convulsions after operation and feels considerably stronger.

Case A241430. Mr. H. H. W. H., aged 31, came to the clinic August 7, 1917, with a history of the onset of headaches in August, 1916, and shortly thereafter numbness of the left foot which gradually extended upwards. He fell and a convulsion followed. His left leg and arm were cramped badly. He bit his tongue and frothed at the mouth. These

spells occurred during the day and lasted about fifteen minutes. The symptoms were always the same, and the attacks became more frequent, sometimes coming every day.

The patient's grandmother was insane and his father a chronic alcoholic. The patient did not take stimulants. His mother and one brother were subject to severe sick headaches. He had two healthy children. He had had neisserian infections at the age of fourteen, sixteen and twenty-three. Since December 2, 1917, the patient had not worked, because he felt too weak and had the spells almost daily. There was a tinnitus in his right ear. He had taken bromids for the past few weeks and had not had an attack.

Physical examination: The patient was well nourished, weighed 132 pounds, and was 5 feet 6 inches tall. His speech was a little slow; the Romberg was positive. He had bilateral sensitive areas in both lower abdominal quadrants. His movements were a little unsteady and he responded slowly. His fundi were normal. The routine examinations of urine and blood, and the blood pressure tests were all normal. A test meal showed free hydrochloric acid of 68 and combined acid of 14. The blood Wassermann test and the spinal fluid Wassermann test in dilutions of 0.2, 0.3, 0.4 and 0.5 cc., the Nonne test were negative. There was one small lymphocyte. The roentgenogram of the stomach was negative. A roentgenogram of the head showed a calcified area in the right parietal area. The temperature was normal.

Neurologic examination (August 7, 1918): The neurologic examination was negative with the exception of nystagmus. There was a very slight diminution in tactile sensibility over the left foot, a marked decrease in joint sensibility of the left toes, a very definitely increased tendo achilles reflex on the left and bilateral absence of the abdominal reflexes. A roentgenogram of the head showed a calcified area in the right parietal region. The spinal fluid examinations were negative.

A diagnosis of migraine and Jacksonian epilepsy, due to a cortical lesion involving the foot center of the left rolandic area, was made and exploration advised.

Barany test: A Barany test was made and showed a spontaneous vertical nystagmus with the patient looking up and a nystagmus down on looking downward (Chart 4). A spon-

taneous pointing fault was present with the left arm. The turning revealed an exaggeration of vertigo. The caloric test presented a perverted nystagmus on stimulation of the right and left vertical canals. Also all verticals gave marked vertigo and a pointing fault for the right arm. The horizontal canals showed a pointing fault with the right arm. The perverted nystagmus obtained in this case was due to an interference in the posterior longitudinal bundles. This, like almost all similar reactions, shows a partial loss of function rather than a total absence, which is typical of the responses due to pressure. There was also a pointing fault for the right arm from all canals. At first glance, this would seem to indicate a cerebellar lesion, because the synergetic movements are controlled by this part of the brain. Such an assumption was wrong in this case for two reasons: first, because the pointing fault was only with the right arm, and second, because stimuli do get through and produce normal past pointing. This fault, therefore, is not due to a cerebellar lesion, but to an interference with the tracts leading to the hindbrain. Such an interference is explained by the effect of pressure on the middle cerebellar peduncles.

In this case the reaction consisted of exaggerated vertigo throughout all tests, perverted nystagmus from all vertical canals and a pointing fault for the right arm from all the semicircular canals. A Barany diagnosis was therefore made of a supratentorial lesion causing considerable pressure downward on the peduncles, stem, and posterior longitudinal bundles. It was impossible to designate the side.

August 22, 1918, a large right subtemporal decompression was made. The cortex was opened and a diffuse, firm tumor was found in the sensory area of the right hemisphere. It was not encapsulated. As it could not be removed, 50 mg. of radium were inserted. The tumor was firm and contained calcium.

Case A221019. Mr. J. A. M., aged 46, came to the clinic February 5, 1918. He had been rather nervous, especially during the past two years. He slept well. He used no stimulant except tea once a day. He had one healthy child, aged 17. When the patient was 23 he was injured in a railroad wreck, receiving three wounds on his head. He was uncon-

scious for three hours and was in bed for three or four weeks. November 7, 1916, a tumor was removed from the back of his right thigh. The tumor was not examined pathologically nor preserved. December 9, 1917, a second operation was performed in the scar of the former operation, the details of which are uncertain, and a rectal operation performed for epilepsy.

The patient's clinical history centered around his epileptic spells. The first attack occurred October 21, 1917, the first premonition being temporary blindness, followed by twitching of the eyes. The patient cried out and fell over unconscious, in a generalized convulsion, accompanied by vomiting and frothing at the mouth. He was unconscious for one and one-half hours and awoke very tired and nervous. He had had about fifteen attacks in the last three months; the last one was ten days before examination. The attacks had become more frequent but less severe. The patient complained of gastric symptoms with belching of gas. About January 1, 1918, he had severe colic and upper abdominal pain, with vomiting for one and one-half hours. His bowels were regular and his appetite good. He had had almost constant severe headaches in the back of his head and behind the right ear, but frequently diffuse and bilateral since the onset of trouble. His vision was faulty, and he often found it difficult to touch his nose with his finger.

Physical examination: The patient walked to the clinic unaided. He was 5 feet and 9 inches tall and weighed 171 pounds; four months before he had weighed 200 pounds. There was a scar on his right thigh posteriorly with no signs of recurrence of the former tumor, which probably was a fatty tumor. The rectal and abdominal examinations were negative. He had a definite pyorrhea alveolaris. The systolic blood pressure was 108, and the diastolic 76. A twelve-hour specimen of 350 cc. of urine was acid in reaction and showed a specific gravity of 1021. All other macroscopic and microscopic tests were normal. Hemoglobin was 70 per cent. The red blood count was 4,448,000 and the white 9,400. The blood Wassermann test and the spinal fluid test in dilutions of 0.2, 0.3, 0.4 and 0.5 cc., using Naguche and Melange antigen, were negative. There were seven small lymphocytes in the spinal

fluid. The Nonne test was negative. The blood cholesterol estimated in milligrams for 100 cc. by the Bloor I method was 0.400, and by the Bloor II method 0.444, a difference of 0.044. Roentgenograms of the head and chest were also negative. The temperature was 98.6. Examinations showed the ear, nose nasopharynx, throat, larynx and auditory acuity to be normal.

Neurologic examination (February 5, 1918): Both pupils were somewhat irregular. A slight nystagmus was noted. Vision was normal in both eyes, the fields showed no contraction, and the fundi did not reveal any evidence of pressure. The cranial nerves showed no abnormality, except that on smiling the patient wrinkled the left side of the face somewhat more than the right. The grip on the left, as tested with the dynamometer, registered 20, and on the right, 26. The power of the other muscles of the left arm and left leg was likewise very slightly less than that of the right. The tendon reflexes were somewhat more active on the left than on the right. The plantar response was flexor on both sides. Sensation was normal throughout. Coordination was normal on the right, but slightly impaired on the left. There was a very slight static tremor in the hands. Speech was slurring. The patient was considerably demented, so that cooperation was unsatisfactory. His handwriting showed no changes of significance. The spinal fluid reactions were normal.

The diagnosis was uncertain; however, exploration of the right motor area of the brain for tumor seemed justified.

Barany test: The Barany test (Chart 5) showed no spontaneous pointing faults, normal hearing, and no spontaneous nystagmus. The turning tests were normal except that the vertigo was unusually marked and accompanied by much nausea and retching. The caloric test revealed a rotatory nystagmus to the left of moderate amplitude on stimulation of the right vertical canals in thirty seconds. The pointing and falling tests were increased over normal. The responses from the right horizontal canal were increased, following the onset of a horizontal nystagmus to the left. On stimulation of the left vertical canals there was a rotatory nystagmus to the right of moderate amplitude in forty seconds. All tests and responses from these canals were exaggerated. Stimulation of

the left horizontal canal produced a horizontal nystagmus to the left of moderate amplitude with the accompanying responses exaggerated.

The Barany test in this case is characterized by a generalized exaggeration of all responses without any reactions being absent or diminished. A diagnosis, from the Barany test, of a supratentorial lesion was made. It was impossible in this case to name the hemisphere. After a consultation with the neurologists, a final diagnosis of a tumor of the brain in the right motor area was made and exploration advised.

At operation, February 25, 1918, a nodular and moderately vascular tumor about 6 cm. in diameter was removed by blunt dissection from the right frontomotor area.

Case A246341. Miss D. D., aged 17, came to the clinic September 21, 1918, complaining chiefly of blindness and headaches. The patient entered the clinic walking, but directed by her father. Her grandfather and aunt on her mother's side died of tuberculosis; one aunt died of cancer. The grandmother and two sisters on the father's side died of tuberculosis. The patient since nursing at the breast had had attacks simulating petit mal, typhoid fever at six years of age, malaria in 1917, and repeated rheumatic attacks. Her menses began at twelve and were painful. In February, 1918, she had attacks of pain in the eyes and noticed that her vision was getting poor. Photophobia was marked. The patient frequently vomited when the headaches were severe, but the vomiting was not of the projectile type. She was relieved of this by lying down and closing her eyes. Sight began rapidly to disappear and was gone in ten days. It disappeared first in the right eye, which had pained her most. At the time of examination she could see only bright lights at night, and petit mal attacks were occurring two or three times daily.

Physical examination: The patient, whose family is inclined to obesity, weighed 227 pounds and was 5 feet and 6 inches in height. The examination revealed few abnormalities. Her entire abdomen was tender to touch. There was doubtful light perception in the right eye. The discs had swollen margins with complete secondary atrophy of the nerve heads. The vitreous contained many small stringy floating opacities from a low grade uveitis. The blood Wassermann

tests in dilutions of 0.3 and 0.4 cc. were negative. The vaginal smear showed gonococci. There was a trace of albuminuria. The white blood cell count was 14,000. The other routine tests were normal. A roentgenogram of the head showed a calcified area in the right temporal region with evidence of intracranial pressure.

Neurologic examination (September 21, 1918): On neurologic examination a complete blindness from secondary optic atrophy, with a divergent strabismus, was noted. The pupils were dilated and did not respond to light. The patellar reflexes could be obtained only on reinforcement, and the tendo Achillis responses were considerably diminished. The plantar reflexes were uncertain but probably flexor.

A diagnosis of probable brain tumor, without definite localizing signs, was made.

Barany test: In carrying out the Barany test (Chart 6) turning was not done because of the patient's general physical condition. There was spontaneous bilateral horizontal nystagmus. The hearing test showed the high fork and the Schwabach test slightly reduced. The pointing was normal. In analyzing the caloric test we noted a prompt response in the nystagmus from all vertical canals. Vertigo was exaggerated in both instances, but much more on the left side than on the right. The pointing tests were questionable chiefly because the patient complained of vertigo and her cooperation was poor. However, a response with the left hand came through on stimulation of the right horizontal canal, and a lesion of the cerebellum was thereby eliminated. When the left canals were tested there was a tendency to hold the eyes to the left. The right cerebellum was considered normal, because a normal reaction was obtained. The Barany test showed, therefore, prompt nystagmus associated with exaggerated vertigo, falling, conjugate deviation of the eyes, and pointing faults, and the diagnosis therefrom was supratentorial tumor causing pressure on the stem and middle cerebellar peduncles.

November 1, 1918, a large subtemporal decompression was performed on the right side. Two and one-half centimeters below the surface a mass was found in the right cerebral hemisphere which contained yellow mucilaginous fluid. The tumor

was not encapsulated and hence enucleation was not attempted. The patient recovered from the operation and went home.

Case A224782. E. S., a girl, aged 7, was brought to the clinic March 11, 1918. The child had had a bilateral frontal headache August, 1917, and had appeared dazed for ten minutes. After two months the headaches were more frequent and severe, and accompanied by projectile vomiting. The patient walked into the clinic without aid. She was the second child of a large family. She had had scarlatina at 3, pertussis at 5, measles and chickenpox at 7. A choked disc had been diagnosed in December, 1917.

Physical examination: The physical findings and laboratory tests were negative. The temperature was normal. Blood Wassermann tests on the child and parents were negative. The von Pirquet test was normal. A roentgenogram of the head did not aid in the diagnosis.

Neurologic examination (March 11, 1918): The neurologic examination showed the pupils to be moderately dilated; reaction to light was normal on the left, but on the right was followed by a secondary dilatation; vision in the right was 20/200 and in the left was reduced to the counting of fingers. There were bilateral choked discs. The mental condition was normal. Otherwise the findings were negative throughout. July 15, 1918, the patient developed a marked nystagmus, described in detail on the Barany sheet.

Barany test: The Barany test (Chart 7) revealed active nystagmus on turning, with absence of vertigo. The caloric test of the right canals showed an absence of vertigo and falling. On the left, however, an absence of nystagmus was obtained from the vertical canals accompanied by the absence of vertigo and falling. There was also a pointing fault for both arms. The right horizontal tract responded in a normal manner. A diagnosis was made of pressure from above on the midstem region, especially on the left, accompanied by a disturbance in the cerebellar tracts in the middle cerebellar peduncle on the left. A summary of the entire case revealed the pressure of an intracranial lesion without localizing signs. On account of the few findings it was decided to do a lumbar puncture every three to five days, withdrawing 8 to 15 cc. of

fluid, and within a few hours to examine the fundi. This therapy had little effect and the choked discs did not change.

A right subtemporal decompression and exploration were done April 1, 1918; no tumor was found. The patient left the clinic but did not improve.

September 19, 1918, the patient returned. At this time she inclined toward the left in walking or sitting. She had involuntaries on awakening in the morning and on arising had a tendency to fall backward. She had tinnitus in the right ear. No loss of motion was noted, but she felt "dopey" all that day.

Neurologic examination (Sept. 19, 1918): The optic atrophy and loss of vision were complete. The pupils were dilated and reacted only to accommodation. Marked nystagmus was still present. Speed of voluntary movements was moderately reduced over the entire right side, although the co-ordination tests were practically normal. A tremor of moderate rate and small amplitude developed on movement. The plantar reflex by the Babinski method was extensive on the right and doubtful on the left. At times, particularly in the morning, there was a tendency to urinary incontinence. Percussion of the head did not reveal local tenderness and imparted a cracked pot resonance. No other significant abnormalities were noted.

A diagnosis of brain tumor was made; however, localization was by no means certain. The signs suggested a cerebellar involvement.

Barany test: A second Barany test (Chart 8) was made which presented very definite changes from the first. There were now a spontaneous bilateral horizontal nystagmus and vertical nystagmus on looking up. A spontaneous pointing fault with the left arm was also found. The right canals, on turning, gave a perverted nystagmus composed of a rotatory and horizontal nystagmus accompanied by a vertical nystagmus. There were also conjugate deviation of the eyes and falling to the right, without subjective vertigo. By stimulating the right vertical canals with cold water, a perverted nystagmus occurred in fifty seconds, consisting of a vertical and a left rotatory nystagmus. The stimulation of the horizontal canals produced a rotatory nystagmus to the left and conjugate deviation. There were also pointing faults with both hands. When the left verticals were similarly stimulated a perverted nys-

tagmus began almost at once, consisting of a vertical, horizontal, and rotatory nystagmus to the left. There was no nystagmus found on stimulation of the horizontal canal. There was no subjective vertigo or falling. A pointing fault with both hands occurred when the horizontal canal was activated.

A diagnosis, from the Barany test, of a lesion in the right cerebral hemisphere, causing great pressure downward, was made.

The second Barany test shows very definitely the effect of prolonged and increased pressure on the stem, posterior longitudinal bundles, and on the middle cerebellar peduncles. The pressure was more intense on the right side, as evidenced by the perverted nystagmus and conjugate deviation which occurred on the right side only. This effect was also evidenced by the fact that the middle cerebellar peduncle on the right was interfered with to the extent that stimuli did not get through to the cerebellum. Evidences of marked pressure signs appeared through both Barany tests, especially the second.

After considerable deliberation a simple cerebellar decompression was performed September 20, 1918. The exploration revealed nothing. The patient died September 22, 1918, of a bilateral lobar pneumonia. Necropsy revealed a glioma of the right frontal lobe measuring 6 by 8 by 5 cm., just anterior to the explored area (Figs. 4 and 5).

Case A248887. Mr. E. B., aged 32, came to the clinic October 16, 1918. The patient's health had been good until March, 1918, when he began to have severe occipital headaches, more marked on the right side, often accompanied by vomiting. In April, 1918, weakness developed in the left arm and extended to the left leg. The weakness gradually increased until June, and the left side of the face became paralyzed. The patient had been a steady alcoholic until March, 1918. The family history was not important.

Physical examination: The physical findings were negative. The blood pressure, urine, and blood were normal. The blood Wassermann test and the spinal fluid Wassermann test with 0.3 and 0.4 cc. were negative. There was one lymphocyte. The Nonne was negative. There was no choked disc. The

roentgenogram showed a very large sella and apparently a calcified pineal body.

Neurologic examination (October 19, 1918): Record was made of normal vision, an active bilateral neuroretinitis, a spontaneous nystagmus, a slight inequality in the size of the pupils, the left being somewhat larger than the right, and a pupillary reaction which was slow but of good amplitude. Rotation of the eyeballs upward was limited. The muscles supplied by the middle and lower divisions of the left facial nerve were slightly but very definitely weak. The left arm and leg showed a moderate weakness, with a corresponding diminution in speed and increase in tonus. Coordination in the left arm was somewhat impaired. The thigh-trunk sign of Babinski was positive on the left. The gait was that of a patient with a left sided hemiplegia. Touch and pain sensibilities were slightly diminished over the entire left side and moderately active on the right; the abdominal reflexes on the left were absent and on the right diminished; the plantar responses were normal. Attention and cooperation were very poor. The spinal fluid findings were negative.

The neurologic diagnosis was tumor cerebri of the right frontomotor area, probably subcortical and diffuse.

Barany test: A Barany test (Chart 9) showed a generalized hyperactivity of responses throughout, except the nystagmus from the right verticals. On turning, the responses of nystagmus were of decreased duration. The caloric test showed an absence of nystagmus on stimulation of the right vertical canals for three minutes; this was accounted for on the basis of pressure from above. The effect of pressure from above explains the absence of falling and the slightly reduced vertigo. The left vertical canals presented normal nystagmus time, but vertigo was markedly increased and associated with vomiting, thus giving evidence of exaggerated responses. Falling was to the left. Pointing was normal for all canals with the right arm, but absent with the left, due to the hemiplegia. Exaggerated responses, especially on the left, with pressure effect on the left brain stem, were present. This, in the presence of a definite hemiplegia, made the diagnosis absolute. The Barany report was the presence of a supratentorial lesion in the right cerebral hemisphere.

October 26, 1918, a cyst was removed from the right frontomotor area. The microscopic diagnosis showed the tissue to be that of a smooth walled cyst (Fig. 6).

Case A308888. N. K., a boy aged 10, was admitted to the clinic March 16, 1920. The patient had been well until October, 1918, when he developed a headache, followed by right side convulsions which continued several hours until chloroform was given. Occasional spells of unconsciousness occurred every week or two. In March, 1919, a blood test was made, and several salvarsan injections were given, followed by iodids and bromids. The child was entirely free from trouble for two months in the fall of 1919. In December he again had attacks of headaches, followed by unconsciousness. About six weeks before examination, pain in the right leg developed which lasted several hours. In February, 1920, a definite weakness of the right arm and leg and a limp were noticed, and the convulsions became more frequent and severe, with twitching of the right side of the face. The child had been unable to walk for the past week. He had had two tonsil and adenoid operations.

Neurologic examination (March 16, 1920): Choked discs of three diopters on the left and of two diopters on the right, with normal vision, were disclosed. There was a slight ptosis on the left. Muscular movements of the forehead and about the mouth on the right were diminished in strength. There was a marked right side hemiplegia. The tendon reflexes on the right were more active than those on the left; the superficial reflexes were normal. The patient had a very marked motor aphasia.

The neurologic diagnosis was tumor involving the left precentral area.

Barany test: The Barany test (Chart 10) revealed normal hearing and no spontaneous nystagmus. The caloric test of the right vertical canals showed a mixed type of rotatory and horizontal nystagmus to the left, of wide amplitude. Vertigo was exaggerated, as were the pointing tests for the left arm. The hemiplegia made all pointing tests with the left arm impossible. The right horizontal canal showed a horizontal nystagmus to the left of wide amplitude, with exaggerated past-pointing with the left arm. The stimulation

of the left vertical canals with cold water revealed a rotatory nystagmus to the right of wide amplitude and of very prompt onset. Vertigo and past pointing were exaggerated. Stimulation of the left horizontal canal revealed increased vertigo and past pointing. The eyes remained turned to the left.

A Barany diagnosis was made of a supratentorial tumor in the left hemisphere.

The patient was operated on March 15, 1920, and a tumor measuring 5 by 4 by 3 cm. was partially removed from the surface of the left temporoparietal lobe, posterior to the fissure of Rolando. The tumor was not encapsulated and it was impossible to remove it entirely. The pathologist reported a glioma. (Fig. 7).

DISCUSSION.

The nine cases of proved tumors above the tentorium, all of which were primarily and independently diagnosed by the Barany test, present many interesting features.

In general there are two large groups of phenomenon syndromes that are typical of supratentorial tumors. The first group shows a very clear and marked response of nystagmus, vertigo, falling and past pointing. If the lesion is near the surface in one cerebral hemisphere, this reaction will be on the side of the lesion. Such unilateral activity of all responses cannot present any difficulty in the localization of the lesion. Symptoms of the second large group, while not so common, are just as typical of cerebral tumor as those of the first group, in which there is very clearly an active response of the nystagmus from one set of canals only, associated with an interference with the tracts in the stem or peduncles, and giving rise to diminished responses or to absence of the responses ordinarily attributed to these areas. In other words, the predominating feature, aside from an early and exaggerated nystagmus, may be the responses due to pressure.

It has been known for some time that the pressure of these tumors frequently presents the only signs and symptoms of an intracranial lesion. Choked disc and enlargement or obliteration of the sella are two notable examples which have little to do with the location of a brain lesion, but which are due to the increased intracranial pressure. So, too, the Barany test may show an apparent predominance of responses ordi-

narily attributed to the stem or peduncles, but which, with an exaggerated nystagmus, are due to pressure. Furthermore, these pressure responses may be on the same side or on the opposite side of the lesion. With an exaggerated and early response of nystagmus, a Barany test may reveal only those responses ordinarily attributed to a fault in the cerebellum, yet this latter response is entirely due to the pressure effects on the tracts leading to the hindbrain. This effect of pressure may be so marked that a pointing fault may be obtained for both arms from all the canals on one side, but, as a rule, by prolonged stimulation it is possible in these cases to get a stimulation through and thus obtain normal past pointing from one or both arms.

It is noteworthy that the Barany test is frequently the only test or group of tests which localizes an intracranial lesion. While this is perfectly true, it is a colossal error for any one doing such tests to be independent of all other factors in the case. It is frequently of the highest value to consult with the neurologist after the Barany diagnosis has been made.

There is another interesting observation, noted in my experience of more than fifty Barany tests, indicating supratentorial lesions, which may explain the absence or predominance of phenomenon syndromes attributed to pressure associated with cerebral lesions in different areas. In lesions on or near the surface of the cerebrum the Barany usually shows all responses exaggerated, with little or no pressure phenomena. On the other hand, lesions deep in the cortex usually show but slightly exaggerated responses, associated with marked reduction of other responses due to pressure. This observation, while very striking, is not conclusive. Many Barany tests can be explained more easily by this theory than by any other. Some of the cases presented here exemplify this observation.

In order to make clear the relative position of the Barany in the tumors localized in the cerebrum, it will only be necessary to state that the diagnosis of the location of an intracranial lesion by this test is considered as only one of a chain of evidences when the final diagnosis is determined by the neurologist. Hence, the final location of such a tumor is made by the neurologist, who considers all symptoms and signs,

including the Barany test. The operative and necropsy findings should also be studied in checking up on Barany responses which apparently do not fit or which at least make the exact localization of a lesion difficult.

CONCLUSIONS

Conclusions drawn from nine proved cases of supratentorial tumors are not, of course, sufficient to account for the relation of the Barany test to lesions above the stem. However, conclusions as follows may be made:

1. A phenomenon syndrome, consisting of exaggerated nystagmus, vertigo, falling and past pointing, suggests a Barany diagnosis of a supratentorial lesion.
2. The more typical reactions may be complicated by responses due to pressure.
3. Variation in the responses may depend on the location of the lesion within the cerebrum.
4. A study of the operative and necropsy findings will be of material aid in correctly interpreting subsequent responses due to a supratentorial tumor.

Fig. 1. (Case 236828.) Photomicrograph of glioma from the right frontomotor area of the brain. x 100.

Fig. 2. (Case A293010.) Endothelioma of brain adherent to dura.

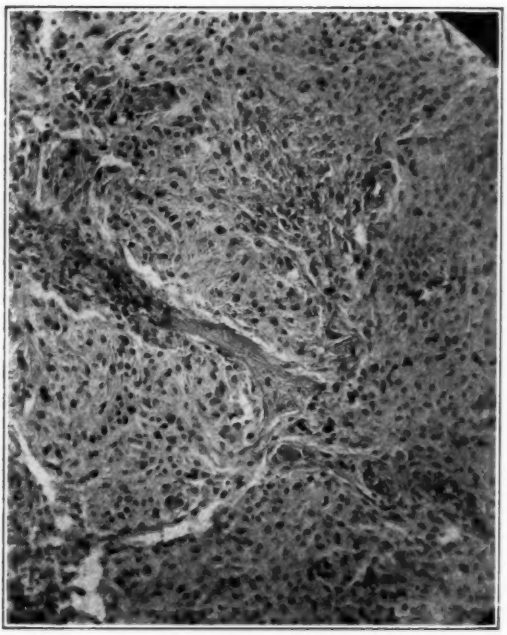


Fig. 3. (Case A293010.) Photomicrograph of section of gross specimen of endothelioma of brain shown in Figure 2. x 100.

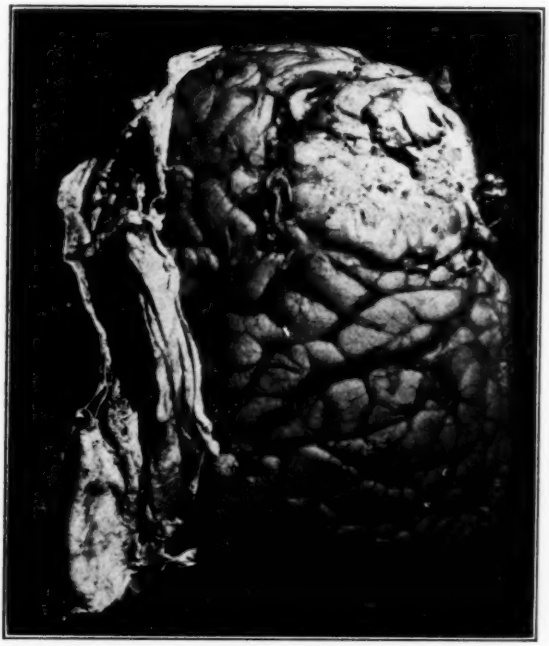


Fig. 4. (Case A224782.) Brain showing glioma of the right frontal lobe.

Fig. 5. (Case A224782.) Photomicrograph of glioma of right frontal lobe of brain shown in Figure 4.

Fig. 6. (Case A248887.) Brain showing area from which smooth walled cyst was removed at operation.

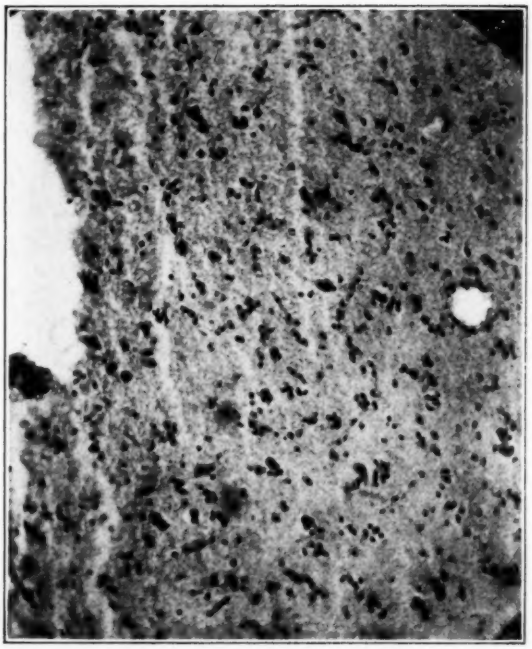


Fig. 7. (Case 308888.) Photomicrograph of glioma removed at operation from left temperoparietal lobe of the brain.

CHART 1. EQUILIBRATORY TESTS

L	HEARING	L
1wv	Noise	1wv
=	Weber	=
—	Rfane	—
N	Schwabach	N
-14	C	-10
- 6	C4	- 6
0	Tinnitus	0
0	Fistula	0
0	7th	0

NYSTAGMUS

Looking to right ←

Looking to left →

Looking up 0

Looking down 0

TO RIGHT

Amp 1 time 24 secs.

→

TO LEFT

Amp 1 time 22 secs.

←

RIGHT

Amp 2 time 25 secs.

HEAD BACK

Amp 3

→

LEFT

Amp 2 time 25 secs.

←

HEAD BACK

Amp 3

←

	SPONTANEOUS	POINTING	
	Shoulder from above	With right	With left
		2 in. to Lt.	2 in. to Rt.
Nystagmus		1 in. to Lt.	T* to
Vertigo		T	
Past pointing			
Falling			
Romberg			
Head to right			
Head to left			
Overthrow			
	TURNING		
Nystag		to	to
Vertigo	26 seconds without vomiting	to	to
Pointing			
Falling			
Nystag		to	to
Vertigo	22 seconds without vomiting	to	to
Pointing			
Falling			
	CALORIC 80° F.		
Nystag	Rapid onset	4 in. to Rt.	3 in. to Rt.
Vertigo	Marked	to	to
Pointing			
Falling	To the right marked onset		
		4 in. to Rt.	4 in. to Rt.
Nystag	Early perverted type	2 in. to Lt.	2 in. to Rt.
Vertigo	Marked with vomiting	T to	2 in. to Lt.
Pointing	Fault with left arm		T
Falling	To left; pointing fault to left		
		4 in. to Lt.	T to

*Touch.

Case 236828

CHART 2. EQUILIBRATORY TESTS

R	HEARING	L
WV	Noise	WV
=	Weber	=
+	Rinne	+
N	Schwabach	N
N	C	N
N	C4	N
0	Tinnitus	0
0	Fistula	0
0	7th	0

Patient was very sick and the turning test was not attempted.

NYSTAGMUS

Looking to right 0

Looking to left 0

Looking up 0

Looking down 0

TO RIGHT

Amp. ..... time .....

TO LEFT

Amp. ..... time .....

RIGHT

Amp. 2 time 1 sec. 30

→

HEAD BACK

Amp. 2

→

LEFT

Amp. ..... time 4 sec.

None

HEAD BACK

Amp. 2

←

SPONTANEOUS

No cooperation on part of patient

Nystagmus ..... to .....

Vertigo .....

Past pointing .....

Falling .....

Romberg .....

Head to right .....

Head to left .....

Overthrow .....

TURNING

Nystag. ..... to .....

Vertigo ..... to .....

Pointing .....

Falling .....

Nystag. ..... to .....

Vertigo ..... to .....

Pointing .....

Falling .....

CALORIC 60° F.

Nystag. Perverted ..... to .....

Vertigo Marked ..... to .....

Pointing Could not be tested .....

Falling To left ..... to .....

Nystag. None from verticals ..... to .....

Vertigo None ..... to .....

Pointing Could not be tested .....

Falling To right ..... to .....

POINTING

With right	With left
to	to
to	to
to	to
to	to
to	to
to	to
to	to
to	to
to	to

Case 235175

CHART 3. EQUILIBRATORY TESTS

R	HEARING	L
WV	Noise	WV
N	Weber	N
N	Rinne	N
N	Schwabach	N
N	C	N
N	C4	N
+	Tinnitus	+
0	Fistula	0
0	7th	0

NYSTAGMUS

Looking to right ←

Looking to left →

Looking up ↑

Looking down ↓

TO RIGHT

Amp. ..... time .....

TO LEFT

Amp. ..... time .....

RIGHT

Amp. 1 time 2 sec. 40

HEAD BACK

Amp. 2

→

LEFT

Amp. 2 time sec. 22

HEAD BACK

Amp. 2

←

SPONTANEOUS

	Shoulder from above	POINTING: With right	POINTING: With left
Nystagmus		T ... to ...	T ... to ...
Vertigo		... to ...	... to ...
Past pointing			
Falling			
Romberg			
Head to right			
Head to left			
Overthrow			

TURNING

Nystag.		... to ...	... to ...
Vertigo		... to ...	... to ...
Pointing			
Falling			

Nystag.		... to ...	... to ...
Vertigo		... to ...	... to ...
Pointing			
Falling			

CALORIC 62o F.

Nystag.	Delayed	T ... to ...	1in. to Rt.
Vertigo	Present with set pauses	1in. to Rt.	1in. to Rt.
Pointing	Reduced reaction		
Falling	Not present		
	Pointing increased	16in. to Rt.	10in. to Rt.

Nystag.	Very active onset	14in. to Lt.	16in. to Lt.
Vertigo	Marked with vomiting	... to ...	... to ...
Pointing	Greatly increased		
Falling	Marked to the left		
	Very active reactions	14in. to Lt.	14in. to Lt.

Case 293010

CHART 4. EQUILIBRATORY TESTS

R	HEARING	L
....	Noise	....
....	Weber	....
N	Rinne	N
N	Schwabach	N
N	C	N
....	C4	....
+	Tinnitus	+
....	Fistula	....
....	7th	....

NYSTAGMUS

Looking to right....0

Looking to left....0

Looking up....↑

Looking down....↓

TO RIGHT

Amp....1....time....25 sec.

→

TO LEFT

Amp....1....time....22 sec.

←

RIGHT

Amp....1....time....50 sec.

↗ HEAD BACK ↑

Amp....2

→

LEFT

Amp....1....time....50 sec.

↑ HEAD BACK ↖

Amp....2

←

SPONTANEOUS

Shoulder from above

Nystagmus....

Vertigo....

Past pointing....

Falling....

Romberg....

Head to right....

Head to left....

Overthrow....

TURNING

Nystag....

Vertigo....Marked

Pointing....

Falling....

Nystag....

Vertigo....Marked

Pointing....

Falling....

CALORIC 68° F.

Nystag....Perverted for vertical

Vertigo....Marked

Pointing....Fault for right arm

Falling....Normal

Point fault for right arm

Nystag....Perverted

Vertigo....Marked

Pointing....Fault with right arm

Falling....Normal

Pointing fault with right arm

POINTING

	With right	With left
	1in. to Rt.	T to
Nystagmus	to	to
Vertigo		
Past pointing		
Falling		
Romberg		
Head to right		
Head to left		
Overthrow		
Nystag	to	to
Vertigo	to	to
Pointing		
Falling		
Nystag	to	to
Vertigo	to	to
Pointing		
Falling		
Nystag	T to	2in. to Rt.
Vertigo	to	to
Pointing		
Falling	T to	2in. to Rt.
Nystag	T to	4in. to Lt.
Vertigo	to	to
Pointing		
Falling	T to	4in. to Lt.

Case 241430

CHART 5. EQUILIBRATORY TESTS

R	HEARING	L
WV	Noise	WV
=	Weber	=
....	Rinne	....
N	Schwabach	N
N	C	N
N	C4	N
0	Tinnitus	0
0	Fistula	0
N	7th	N

NYSTAGMUS

Looking to right 0
Looking to left 0
Looking up C
Looking down C

TO RIGHT

Amp. 2 time 26 sec.

TO LEFT

Amp. 2 time 26 sec.

RIGHT

Amp. 2 time 30 sec.

Amp

LEFT

Amp. 2 time 40 sec.

Amp

SPONTANEOUS	Shoulder from above	POINTING: With right	POINTING: With left
		T to	T to
Nystagmus		to	to
Vertigo			
Past pointing			
Falling			
Romberg			
Head to right			
Head to left			
Overthrow			
TURNING			
Nystag.	Marked and pure	to	to
Vertigo.	Marked nausea and retching	to	to
Pointing	Normal		
Falling.	Slight to right		
Nystag.	Marked and pure	to	to
Vertigo.	Marked nausea and retching	to	to
Pointing	Normal		
Falling.	Slight to left		
CALORIC 68° F.			
Nystag.	Marked	12in. Rt.	10in. Lt.
Vertigo.	Increased from normal	to	to
Pointing	Exaggerated		
Falling.	Very marked		
	Responses increased	8 in. Rt.	6in. to Lt.
Nystag.	Moderate amplitude	8 in. Lt.	6in. to Lt.
Vertigo.	Increased	to	to
Pointing	Exaggerated		
Falling	Present		
	Responses exaggerated	6 in. Lt.	6in. to Lt.

Case 221019

CHART 6. EQUILIBRATORY TESTS

R	HEARING	L
WV	Noise	WV
=	Weber	=
+	Rinne	+
-2	Schwabach	-2
N	C	N
-6	C4	-6
0	Tinnitus	0
0	Fistula	0
0	7th	0

NYSTAGMUS

Looking to right. ←
Looking to left. →
Looking up. 0
Looking down. 0

TO RIGHT

Amp. ..... time. .....

TO LEFT

Amp. ..... time. .....

RIGHT

Amp. 1 time. 40 secs.

HEAD BACK

Amp. 2

LEFT

Amp. 2 time. 40 secs.

HEAD BACK

Amp. 2

SPONTANEOUS

	Shoulder from above
Nystagmus	
Vertigo	
Past pointing	
Falling	
Romberg	
Head to right	
Head to left	
Overthrow	

TURNING

Nystag.	
Vertigo	
Pointing	
Falling	

CALORIC 60° F.

Nystag.	Prompt onset
Vertigo.	Not exaggerated only
Pointing.	Questionable
Falling.	Not induced
	Questionable pointing fault
Nystag.	Prompt onset
Vertigo.	Marked with nausea
Pointing.	O.K.
Falling.	To left
	Normal
	Tendency for her to hold eyes to left.

POINTING

	With right	With left
	T ... to ...	T ... to ...
Nystagmus	... to ...	... to ...
Vertigo		
Past pointing		
Falling		
Romberg		
Head to right		
Head to left		
Overthrow		
Nystag.	... to ...	... to ...
Vertigo	... to ...	... to ...
Pointing		
Falling		
Nystag.	... to ...	... to ...
Vertigo	... to ...	... to ...
Pointing		
Falling		
	T ... to ...	... to ...
Nystag.	6 in. Lt.	8 in. to Lt.
Vertigo	... to ...	... to ...
Pointing		
Falling		
	6 in. Lt.	4 in. to Lt.

Case 246431

CHART 7. EQUILIBRATORY TESTS

R	HEARING	L
....	Noise	....
....	Weber	....
....	Rinne	....
N	Schwabach	N
N	C	N
N	C4	N
....	Tinnitus	....
....	Fistula	....
....	7th	....

First Barany.

NYSTAGMUS

Looking to right ... 0
Looking to left ... 0
Looking up ... 0
Looking down ... 0

TO RIGHT

Amp. 1 time 15 sec.

→

TO LEFT

Amp. 1 time 16 sec.

←

RIGHT

Amp. 1 time 1 sec.

HEAD BACK

Amp 1

→

LEFT

Amp. ... time 4 sec.

Only jerks

HEAD BACK

Amp ...

←

SPONTANEOUS

	Shoulder from above
Nystagmus	
Vertigo	
Past pointing	
Falling	
Romberg	
Head to right	
Head to left	
Overthrow	

TURNING

Nystag.	Rapid onset
Vertigo	Absent
Pointing	
Falling	
Nystag.	
Vertigo	Absent
Pointing	
Falling	

CALORIC 68° F.

Nystag.	Normal
Vertigo	None
Pointing	Normal
Falling	Not present
	Normal
Nystag.	None in 4 minutes
Vertigo	Absent
Pointing	Fault with both arms
Falling	Not present
	Normal

POINTING

	With right	With left
	T to ...	T to ...
Nystagmus	to ...	to ...
Vertigo		
Past pointing		
Falling		
Romberg		
Head to right		
Head to left		
Overthrow		
Nystag.	to ...	to ...
Vertigo	to ...	to ...
Pointing		
Falling		
Nystag.	to ...	to ...
Vertigo	to ...	to ...
Pointing		
Falling		
Nystag.	2 in. to Rt.	2 in. to Rt.
Vertigo	to ...	to ...
Pointing		
Falling	2 in. to Rt.	2 in. to Rt.
Nystag.	T to ...	T to ...
Vertigo	to ...	to ...
Pointing		
Falling	4 in. to Lt.	3 in. to Lt.

Case 224782

CHART 8. EQUILIBRATORY TESTS

R	HEARING	L
....	Noise	....
....	Weber	....
....	Rinne	....
N	Schwabach	N
N	C	N
N	C4	N
....	Tinnitus	....
....	Fistula	....
....	7th	....

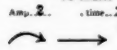
Second Barany.

NYSTAGMUS

Looking to right.....
Looking to left.....
Looking up.....
Looking down.....0

TO RIGHT

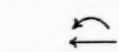
Amp. 2 time 30 sec.



Conjugate deviation

TO LEFT

Amp..... time 25 sec.

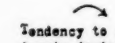


RIGHT

Amp. 2 time 50 secs.

HEAD BACK

Amp.....



Tendency to conjugate deviation

LEFT

Amp. 2 time..... sec.

HEAD BACK

Amp.....

None

SPONTANEOUS

Shoulder from above

Nystagmus.....
Vertigo.....
Past pointing.....
Falling.....
Romberg.....
Head to right.....
Head to left.....
Overthrow.....

TURNING

Nystag. Right rotary and horizontal also vertical
Vertigo. None
Pointing
Falling. To right

CALORIC 60° F.

Nystag. Perverted
Vertigo. None
Pointing. Faults with both arms
Falling. None

Nystag. Perverted
Vertigo. None
Pointing. Reduced
Falling. None

Pointing faults with both arms

POINTING

With right	With left
2 in. to Lt.	T. to
..... to	..... to .....
.....	.....
.....	.....
.....	.....
.....	.....
.....	.....
.....	.....
.....	.....
.....	.....
.....	.....
T. to	T. to .....
..... to	..... to .....
.....	.....
.....	.....
T. to	T. to .....
2 in. Lt. to	1 in. Lt. to .....
..... to	..... to .....
.....	.....
.....	.....
T. to	T. to .....

Case 224782

CHART 9. EQUILIBRATORY TESTS

R	HEARING	L
WV	Noise	WV
=	Weber	=
+	Rinne	+
-5	Schwabach	-5
N	C	N
N	C4	N
+	Tinnitus	+
0	Fistula	0
0	7th	-

NYSTAGMUS

Looking to right 0
Looking to left 0
Looking up 0
Looking down 0

TO RIGHT

Amp. 3 time 16 sec.

TO LEFT

Amp. 2 time 17 sec.

RIGHT

Amp. 0 time 3 sec.

0

HEAD BACK

Amp 1

LEFT

Amp. 1 time 1 = 00

HEAD BACK

Amp 2

SPONTANEOUS

	Shoulder from above
Nystagmus	
Vertigo	
Past pointing	
Falling	
Romberg	
Head to right	
Head to left	
Overthrow	

TURNING

Nystag.	
Vertigo	17 sec.
Pointing	
Falling	
Nystag.	
Vertigo	17 sec.
Pointing	
Falling	

CALORIC 660 F.

Nystag.	Absent	4 in. to Rt.	
Vertigo	Present		
Pointing	O.K. with right		
Falling	Not present		
		4 in. to Rt.	
Nystag.	Hyperactive	6 in. to Lt.	
Vertigo	Marked with vomiting		
Pointing	O.K. with right		
Falling	To left		
	Hyperactive	8 in. to Lt.	

POINTING

With right	With left
T	
	Paralysis

Case 248887

CHART 10. EQUILIBRATORY TESTS

R	HEARING	L
WV	Noise	WV
N	Weber	N
N	Rinne	N
....	Schwabach	....
N	C	N
0	C4	0
0	Tinnitus	0
0	Fistula	0
....	7th	....

Unable to turn eyes upwards or to right.

NYSTAGMUS	SPONTANEOUS	POINTING	
		With right	With left
Looking to right 0	Shoulder from above	2 in. to.	to Lt.
Looking to left 0	Nystagmus	to.	to.
Looking up 0	Vertigo		
Looking down 0	Past pointing		
	Falling		
	Romberg		
	Head to right		
	Head to left		
	Overthrow		
TO RIGHT	**TURNING**		
Amp. 1 time 35 sec.	Nystag. Wide amplitude	to.	to.
→	Vertigo. Present	to.	to.
	Pointing		
	Falling		
TO LEFT			
Amp. 1 time 35 sec.	Nystag. Wide amplitude	to.	to.
←	Vertigo. Present	to.	to.
	Pointing		
	Falling		
RIGHT	**CALORIC 68° F.**		
Amp. 2 time 45 sec.	Nystag. Wide amplitude prompt onset	to.	5 in. to. Rt.
↘	Vertigo. Increased over normal	to.	to.
HEAD BACK	Pointing. Absent with right		
→	Falling. Not induced		
Amp. 2	Eyes turned to left throughout	to.	10in. to. Rt.
→			
LEFT			
Amp. 2 time 43 secs.	Nystag. Prompt onset	to.	6in. to. Lt.
↙	Vertigo. Increased over normal	to.	to.
HEAD BACK	Pointing. Absent with right		
Amp. 2	Falling. No induced		
←	Eyes turned to left throughout	to.	8in. to. Lt.

Case 308888

LXXXI.

PRIMARY PERIOSTITIS OF THE MASTOID.*

By Samuel Salinger, M. D.,

Chicago.

Inflammation of the periosteal covering of the mastoid occurring independently of any infection of the tympanic cavity or mastoid cells is a condition not infrequently encountered and one that merits careful consideration.

The earliest recognition of this condition is to be found in a report of three cases by Voltolini,[1] who regarded them as idiopathic localized phlegmons probably due to exposure to cold, and who postulated the following conclusions: "The disease," says he, "is neither of the auricle, the external auditory canal, the tympanic cavity nor the mastoid; it originates from without and not within the ear; it may progress inward but not very deeply." Politzer[2] states that "the disease may involve only a circumscribed area or may take in the entire mastoid cortex," and that "it is characterized by the formation of a hard, usually red, swelling on the mastoid which slopes down at the border, is accompanied by fever and great pain, and is very tender to the touch."

The etiology in many instances is obscure, and while most authors mention trauma or exposure as the principal exciting causes, a variety of other factors have been reported. These are the estrus larvæ,[1] vicarious menstruation,[3] typhoid fever,[4] tuberculosis,[5] syphilis,[6] actinomycosis,[7] breaking down of the small superficial postauricular lymph gland,[8] erpsipelas[9] and osteomyelitis.[10] Others[11 12 13 14] claim that the infection finds its atrium in the meatus at the cartilagoosseous junction of the posterior wall. Jacques and Gault[7] (also Wurdman[9]) cite several cases which ensued some time after a healed suppurative otitis media, and while not claiming that these are, strictly speaking, primary periostitis, they, nevertheless, are to be distinguished from the cases which accompany infection

*Read before the Chicago Laryngological and Otological Society, March 1, 1920.

of the mastoid cells. They state the proposition thus: "Cases may properly be termed primary when they relate to a local trauma or a systemic infection. When, however, they develop after a recently healed otitis media they are more truly an osteomyelitis that is neither primary nor hematogenous but simply isolated. That is, they develop independently with relation to an otitic cause and are capable of independent treatment." Laurens[15] reports a case where following an acute otitis media a collection of pus was found on the mastoid process, though at operation the antrum and mastoid cells proved to be entirely free from disease. He firmly believes that the infection was carried by the blood stream directly to the cortical periosteum. Cases of the latter type, according to our point of view, should be excluded and none considered as truly primary unless their connection with a tympanic or mastoid infection be definitely ruled out.

The disease in many respects resembles the secondary periostitis or mastoid edema which so frequently accompanies an acute mastoiditis or the early stages of a severe suppurative otitis media. The typical case begins rather abruptly with pain referred to the ear or to the region behind the auricle, which may radiate into the neck or toward the occiput, a moderate rise in temperature and the rapid formation of a hard red swelling over the mastoid, which is usually extremely painful to the touch. Occasionally, according to Politzer, "the inflammation may develop slowly and be practically painless." Such cases, says he, "are likely to lead to superficial necrosis of the cortical layer of the mastoid process with exfoliation of small fragments of bone."

The hearing is frequently diminished, owing to a consecutive middle ear catarrh, and when this condition obtains it adds greatly to the difficulty in diagnosis, particularly if the postauricular swelling develops slowly.

In some cases the posterior meatal wall is involved, thus obscuring a view of the tympanic membrane and presenting a picture closely resembling a true mastoiditis.[16] Not all of the cases go on to suppuration,[17 18 19 13 24] resolution in some instances taking place within a few days. In the majority, however, after a period of from three to ten days, fluctuation can be elicited directly behind or above and behind the auricle.

Occasionally the abscess will rupture spontaneously into the auditory canal at the cartilago-osseous junction or through one of the fissures of Santorini. Also it has been known to rupture into the infraauricular space and to follow the sternomastoid muscle into the neck.

The diagnosis as a rule is easy, if the tympanic membrane can be seen and is found to be normal. However, in those cases where the posterior wall of the osseous meatus is found to be swollen and that portion of the tympanic membrane which is visible is seen to be injected, one might not be able to determine with certainty whether a middle ear or mastoid infection is present and responsible for the symptoms. Particularly so, if one happens to be on the lookout for a streptococcus mucosus infection, which is prone to touch the middle ear but lightly and wreak its havoc almost entirely in the mastoid. In this connection it should be borne in mind, as pointed out by Ostman[20] and by Webster,[25] that swelling of the osseous meatus due to mastoid involvement rarely occurs earlier than the second or third week following the onset of an acute suppurative otitis media, whereas if due to a primary periostitis this swelling develops rapidly and may be found at the very onset of the symptoms concurrent with the swelling behind the auricle. A deep seated furuncle of the auditory canal might be mistaken for a true primary periostitis if accompanied by early postauricular swelling. In these cases, however, the pain and meatal swelling always precede the appearance of the external swelling.

These facts were well illustrated in a case which was under my care a year ago. A gentleman about 40 years of age applied for the relief of nasal obstruction due to a markedly deviated septum and a purulent discharge which originated in the sphenoid and posterior ethmoids on the deviated side. A few days before the date set for operation he complained of pain in the ear, on the same side as the nasal pathology. The examination disclosed a swelling above and anterior to the auricle which was quite tender to the touch. The auditory canal contained a small amount of epithelial debris and was partially blocked by a swelling of the superior and posterior osseous walls which obscured a view of the drumhead. Under palliative treatment the symptoms subsided within three days

and the membrane was then seen to be pale, lusterless, somewhat retracted and not inflamed. The membrane on the opposed side had practically the same appearance. The nasal operations were performed and were healing satisfactorily when on the fourth day the swelling above and anterior to the auricle reappeared, accompanied by pain, tenderness, a slight rise in temperature and a swelling of the posterior and superior walls of the osseous meatus. As the symptoms were increasing in severity, we concluded that the affair was primarily of the auditory canal, the drum membrane not being visible, and, therefore, under local anesthesia, made an incision deeply into the swollen meatal wall, but without encountering any pus. The condition, however, improved immediately, the swelling, both internal and external, subsiding rapidly, and the drum membrane being again visible showed nothing more than a slight injection of its epidermal layer. Two days later the symptoms reappeared with increased severity. The external edema extended from in front of the auricle to the temporal region and behind almost to the tip of the mastoid and was much more tender than before. The osseous meatus was also swollen until the canal was almost occluded. Within four days the swelling localized directly behind and above the auricle, lifting it away from the skull, and showing distinct flucation. An incision was then made under local anesthesia behind and close to the auricle, and a large quantity of pus evacuated. The bone was found denuded of its periosteum, but careful search failed to reveal any fistula either into the bone or into the auditory canal. The wound was closed with drainage. The swelling disappeared within a few days and the wound healed completely within ten days.

Cases very similar to the above have been reported by Meniere[11] and doubtless have also been noted by many of the members of the society. The location of the swelling above and anterior to the auricle is not unusual. Gellé[21] has noted types in which the swelling extended as far forward as the external margin of the orbit, as far backward as the occiput and as far downward as the clavicle.

The unusual feature of the case cited was its delayed development after two abortive beginnings.

Another case of the slowly developing type referred to by Politzer and one in which the etiology was obscure occurred in a little girl of four who had been under the care of a dermatologist for pemphigus and who was referred to me because of pain in the ear. There was no history of either trauma or exposure. The examination disclosed a tender swelling behind and above the auricle. The auditory canal was uninvolved and the tympanic membrane appeared duller and more opaque than in the opposite ear. Although it did not appear to be an acute otitis media, the membrane was nevertheless incised because of a possible connection with the external edema. Nothing, however, was obtained but a little thin mucoid secretion. The case was treated by palliative measures while the postauricular swelling developed in size and tenderness. During this period of five days there was only slight spontaneous pain and the temperature never rose above 100° F. There was scarcely any discharge from the ear following the paracentesis, which promptly closed, leaving the drum membrane as originally found. At the end of the fifth day the auricle was pushed forward and downward by the swelling, which was now extremely tender and slightly fluctuating. An incision was made and a large subperiosteal abscess evacuated. The squamomastoid suture was not patent and no fistula could be discovered. However, just above the posterior root of the zygoma, the bone was found to be eroded over an area about the size of a dime. This was thoroughly curetted, and as no fistula or aberrant cells could be discovered, the wound was then closed with drainage. Within two weeks it was entirely healed.

The X-ray findings prior to operation in both of these cases were not of great assistance in the diagnosis, as the denser shadow on the affected side could equally as well have been produced by pus within the mastoid as on its surface.

In conclusion, the points that should be emphasized in connection with the diagnosis are, first, the absence of any recent middle ear infection; second, the preponderance of the local over the constitutional symptoms, and third, the slow development of suppuration. Because of the last named factors it has been advised by most authors[2 13 9 3] that the swelling be incised early, as the pain is thereby alleviated, and frequently

the condition clears up without the formation of pus. To temporize, as was done in the cases here reported, is to invite unnecessary destruction of tissue, in the one case leading to superficial necrosis of the bone, and in cases reported by Burnett,[22] Hotz,[14] Voltolini[1] and others, the pus burrowing its way into the auditory canal or into the tissues of the neck producing troublesome fistulæ.

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Abstracts From Current Literature.

I.—EAR.

Temporozygomatic Paramastoiditis.

Mouret and Seigneurin.
Rev. de laryngol., etc., Paris, 1920—XLI—65.

In a long and valuable article, Mouret sets forth the necessity of performing a complete operation in every case of mastoiditis. Only in this way can hidden and unsuspected areas of disease lying behind healthy bone be discovered. Illustrative of this he reports in summary nine cases where bone lesions were discovered adjacent to the mastoid, which if left uncared for, would have delayed or rendered healing impossible. These adjacent localities he describes as the paramastoid and gives to an inflammation of the cells involved the term "Temporozygomatic Paramastoiditis."

In the majority of cases, the infection from the middle ear and the antrum is propagated to the squamosa and the squamozygomatic lamella, resulting either in superficial or deep edematous reactions or in the formation of deep or superficial temporal abscesses. The infection is carried to the paramastoid by continuity through the Haversian canals or by the lymphatics or blood vessels. This diffusion is particularly favored when the squamozygomatic lamella contains many cells. Analogous to the cribriform area behind the meatus two cribriform zones are found in this region, the suprameatic zone and the deep temporal zone. Their development varies in different individuals. They both enter largely into the transmission of the infection. Frequently, a fistula can be demonstrated; at other times there is a superficial necrotic erosion. In brief, the infection from the tympanum and antrum invades the bone canals or the bone cells from their attacking the internal cortex by necrosis or the external cortex by necrosis, but also by means of the cribriform, suprameatic and deep temporal cribriform zones. As a result of the presence of a bony lamella which separates the internal and external cortex, the infection may advance to the meninges and brain or externally. If it extends externally there can be either a simple osteitis with edema of the temporal muscle or an ostitis with necrosis and a collection of pus in the deep temporal fossa with more or less lardaceous edema of the temporal muscle, even leading to a necrosis in the bone with the formation of a fistula. The symptoms are slight. The chief symptom is trismis when the muscle is involved, and a painful tumefaction and temporopalpebral edema when the involvement is superficial. Systematic exploration of the paramastoid regions is always indicated in the presence of symptoms of disease. There a careful exploration is doubly called for. This includes a prolongation of the usual retroauricular incision and then the examination of the entire area according to a carefully described plan of procedure.

Harris.

Paralysis of the External Motor Oculi Nerve in the Course of an Attack of Acute Suppurative Otitis Media on the Opposite Side.

De Lamothe.

Rev. de laryngol., etc., Paris, 1920—XLI—198.

The author has recently seen such a case in a child of nine, suffering from acute mastoiditis on the right side requiring operation. Following the operation, meningeal symptoms developed, although the lumbar puncture was normal. Ten days after the operation the child suddenly complained of seeing double. Examination showed a paralysis of the sixth nerve on the left side. The child eventually made a complete recovery.

Various cases of this kind in the literature have been examined with a view to the explanation of the cause. Three theories are advanced: the reflex, anatomic and the serous meningitis. The author regards the theory of the reflex origin as untenable, and as the result of a careful study of a number of skulls, dismisses the possibility of anatomic cause. In his judgment, the only possible explanation is that of a serous meningitis. He frankly admits that his theory in itself does not explain why the paralysis should be produced on the other side. In the great majority of cases he admits that the paralysis follows on the side of the diseased ear; on the other hand, it must not be forgotten that if the cause of the hypertension of the cerebrospinal fluid is well localized, the pressure will be exerted not there alone but upon all the cranial nerves. Under these conditions the most fragile nerve will suffer first. Such being the case, there is nothing to exclude the possibility of the motor oculi nerve on the opposite side being more delicate than the nerve on the side of the ear lesion. So far as treatment is concerned, the two indications are, the drainage of the ear, and secondly the combating of the hypertension of the cerebrospinal fluid.

Harris.

An Unusual Case of Lateral Sinus Thrombosis.

Friedman, Joseph, and Greenfield, Samuel D.

Med. Rec., New York, 1920—LXCVIII—943.

This case presents four special features:

1. The infection through the sclerotic mastoid to the sinus took place with unusually great rapidity. The ear symptoms from the date of onset to the operation were only of ten days' duration.

2. The more pronounced symptoms indicating such a severe involvement were entirely lacking. Nothing that could be detected at the time of examination pointed to the possibility of such a serious complication. It should be remembered that this was an ambulatory case of sinus thrombosis.

3. The actual demonstration of pus in the sinus.

4. The absence of bleeding from the jugular bulb after removing all clots from the sigmoid below the knee to the bulb, showed conclusively that the thrombosis must also have extended at least into the inferior petrosal sinus. Nevertheless, nothing developed as a consequence.

Emil Mayer.

Report on Twenty-seven Cases of Jugodigastric Mastoiditis.

Mouret.

Rev. de laryngol., etc., Paris, 1920—XLI—167.

Lesions of the digastric triangle are most difficult to attack. They are the result of infection of the deep subantral zone, and in order to attack them the superficial subantral zone must be entirely removed. They lie deeply seated on the base of the skull. Total removal of the apex is necessary. Approach must be made through the base of the neck, and the posterior belly of the digastric muscle must be detached from its insertion in the digastric triangle which is the seat of the bony lecions in jugodigastric mastoiditis. Access to this deep region is more difficult than to that on the outside of the muscle; more difficult than that on the inside of the apex. The operator too often stops his work on the internal surface of the apex. If he wishes to discover the jugodigastric regions, he must go further. Mouret distinguishes two parts to this triangle, one posteriorly, the region of the bulb, and the anterior, the floor of the deep subantral zone of the mastoid. It is particularly in the anterior part that the disease is apt to be met. In the twenty-seven cases which he has seen he notes eleven fistulas at the summit of the digastric triangle. His explanation of the number of cases that he has seen and the rarity that they are met with by others, is that due to his early experience he has systematically looked for them and so has found them. Harris.

II.—NOSE.

Nasal Deformities Due to the Submucous Operation, Their Prevention and Their Treatment.

Carter, William Wesley.

Med. Rec., New York, 1920—LXCVIII—810.

The course to be adopted in the treatment of the depressed deformities resulting from the submucous operation admits of no discussion. There is only one satisfactory method by which they may be corrected, and that is by the transplantation of bone or cartilage, or preferably by a combination transplant composed of both cartilage and bone. In these cases a transplant from the ninth rib, composed of two-thirds bone and one-third cartilage, is taken at the junction of the rib with the costal cartilage. The transplant is introduced from within the nose and no scar results. The full strength of the nasal bridge is restored and the repair is permanent, for some cases have been under observation long enough to demonstrate this fully. The bone transplant establishes a firm bony union with the frontal bone and supports a nasal bridge in its normal position; the cartilage preserves the normal flexibility of the nasal tip.

While the number of deformities resulting from the submucous operation are few in comparison with the number of cases operated upon, still the knowledge that these deformities are serious in their import and that they may and do occur in the hands of

unskilled operators and others when proper precautions are not observed, if sufficient justification for this presentation of the subject. Emil Mayer.

Etiology of Deviations of the Nasal Septum: Anatomic Theory.

Spielberg, William.

J. Am. M. Ass., Chicago, 1920—LXXV—1646.

The nasal septum divides the bony structure into two compartments. It is surrounded by hard, unyielding, bony tissues which begin to ossify from the first to the eighth week of gestation. The septum, composed of the vomer, perpendicular plate of the ethmoid and triangular cartilage, ossifies so far as the bony portion is concerned, from the second year to puberty. Of course, the cartilage remains soft throughout life.

It is easily conceived, therefore, that the surrounding bony structures, ossifying at a much earlier period than the bony septum, will cause the septal constituents to deflect or deviate in order to permit it to occupy its proper space. It is as if one should attempt to insert a paper perpendicularly into a box of small dimensions. The paper would, of course, assume the form of an S or bend out with concavoconvex configuration. Emil Mayer.

Chronic Maxillary Sinusitis and Albuminuria.

Liebault.

Rev. de laryngol., etc., Paris, 1920—XLI—129.

The author reports four cases of chronic antrum disease (at first unrecognized) which were found to be the cause of a persistent albuminuria which cleared up as a result of treatment to the sinus. He argues, based on the study of these cases, that it must never be forgotten that chronic maxillary sinusitis may be often very atypical, showing itself by only slight nasal secretion, either mucopurulent or with some purulent crusts. In every case it is well to bear in mind, when an individual complains of lowered vitality, is pale, has dyspeptic troubles and has a little albumin in the urine, a careful examination of the sinus with an exploratory puncture will at times give the explanation of the albuminuria and permit the securing of a rapid recovery. Harris

III.—PHARYNX AND MOUTH.

Effect of Tonsillectomy on the Recurrence of Acute Rheumatic Fever and Chorea.

St. Lawrence, William.

J. Am. M. Ass., Chicago, 1920—LXXV—1040.

The writer presents the following conclusions:

1. Eighty-five children, each of whom had presented one or several of the rheumatic manifestations before the tonsils were completely removed, were observed during an average period of three and one-half years after the operation was performed.

2. The tonsils were markedly hypertrophied in 13 per cent of the cases, moderately so in 69 per cent of the cases, and not enlarged in 18 per cent of the cases. They were the site of recurrent inflammation before the tonsils were removed in 73 per cent of the cases. "Sore throat" recurred after removal of the tonsils in 7 per cent of these. At least two operations were necessary before the tonsils were completely removed in 22 per cent of the cases.

3. The tonsillar lymph nodes were enlarged in 100 per cent of the cases before the operation was performed, while in 59 per cent of the cases they were impalpable afterward.

4. One or more acute attacks of rheumatic fever had occurred in forty-two cases before the tonsils were removed. After tonsillectomy there was no recurrence in thirty-five cases, or 84 per cent.

5. One or more attacks of chorea had occurred before the removal of the tonsils in forty cases, and there was no recurrence after the operation in twenty cases, or 50 per cent.

6. Sixty-one cases showed myostitis and bone or joint pains before the operation was performed, and there was no recurrence in forty-seven cases, or 77 per cent.

7. Fifty-eight cases of organic disease of the heart were present in the series. Twelve of these patients had suffered at least one attack of cardiac failure before the tonsils were removed. One patient suffered one attack afterward.

8. The exercise of tolerance in the cases of cardiac disease seemed to be favorably influenced by tonsillectomy in the instances in which indications existed for the removal of the tonsils.

9. Nutrition and general health were improved, and intercurrent disease was less common after the tonsils were removed.

10. Tonsillectomy (complete removal of the tonsils) would seem to be the most important measure at present available for the prevention of acute rheumatic fever and the allied rheumatic manifestations. Emil Mayer.

The Anatomy, Physiology and Pathology of the Tonsillar Structures in Relation to Cryptogenic Infections.

Wood, George B.

Med. Rec., New York, 1920—LXCVIII—595.

It is probable that in the majority of infections of the tonsil that there has been some break in the epithelium due to the necrotic action of the bacterial toxins which have developed from the organisms in the lumen of the crypt. It is possible, however, that a certain amount of toxic material is absorbed directly through normal cryptal epithelium, especially in those cases of chronic tonsillar disease in which the lumen of the crypt has been obliterated, forming within the tonsil a retention cyst of necrotic infected material.

In acute tonsillitis the tonsil is infected in the following manner. The infecting organism gains access to the crypt, producing there, by its growth, sufficient irritation to cause a distinct reaction in the epithelium. This reaction is manifest by a proliferation of the epithelial cells, as shown by the increase of desquamation and of penetration of the basal cells into the tonsil parenchyma and the

presence of mitotic figures in the epithelial cells. If the infecting organism is not of sufficient virulence, this proliferation of the epithelium may prevent invasion, and we have as the result a simple parenchymatous tonsillitis. When the toxic action, however, is more severe, certain portions of the epithelium become necrotic and this process may progress until the parenchyma of the tonsil is opened to the infecting bacteria. The bacteria then passes with the current of the interfollicular tissue toward the lymphatics, or they may become arrested in the germinating follicles where through their development abscess formation takes place in the follicle which later ruptures into a neighboring crypt, producing a purulent discharge at the mouth of the crypt which is so characteristic of the suppurative tonsillitis. The organisms that reach the efferent lymph vessels are carried to the regionary lymph nodes, producing the lymph adenitis which is associated with all severe cases of tonsillitis.

In sumimng up the evidence presented, it would seem that systemic infection, because of the peculiar anatomy of the crypts, and because of the peculiar property of absorbing substances from its crypts, and of the extremely low resisting quality of the cryptal epithelium to bacterial attacks, takes place more readily through the tonsillar structures than through any other portion of the upper respiratory tract. That the tonsil itself is not destroyed by the numerous invasions to which it is subjected must be ascribed to its great power of recuperation and regeneration.

Emil Mayer.

Vincent's Angina Infection—Its. Prevalence, Manifestations, Bacteriology and Treatment—Report of Fifty-six Cases.

Reckord, Frank F. D., Assisted by Baker, M. C.
J. Am. M. Ass., Chicago, 1920—LXXV—1624.

The author desires to emphasize strongly the value of the use of arsphenamin locally in the form of a 10 per cent solution in glycerin, or, for practical purposes, 0.6 gm. of arsphenamin dissolved in two fluid drams of glycerin. It does not seem to disintegrate in this way, and if dissolved in sterile water or salt solution and neutralized with sodium hydroxid, the preparation will become useless in a short while after being prepared. The author believes also that it is better to mix all the powder with the glycerin at one time rather than to dissolve just enough for the particular application; there is no serious objection to this method. The parts to be treated should be thoroughly cleansed and dried, and then the arsphenamin solution applied by a small cotton swab and the material thoroughly rubbed into the lesions. It is surprising to see the good results by applying this treatment twice a day. The duration of the disease is shortened materially, and it seems also to lessen the pain associated, which is particularly prevalent when the gums are involved. It is also indicated in those cases in which the gums are primarily involved and there is infection about the teeth, such as pyorrhea, as the author has found that about 90 per cent of such cases show fusiform bacilli and spirochetes resembling Vincent's organism.

The next substance that gave good results was 2 per cent solution of chromic acid, applied in the same manner or sprayed on with an atomizer. Some observers have used tincture of iodin, colloidal silver, flavine, and a mixture composed of vinum ipecacuanhae, one-half ounce; glycerin, 1 ounce, and liquor arsenicalis, 1 ounce; also powdered methylen blue; but in the author's experience none has given such good results as the solution of arsphenamin.

1. It seems to be definitely proved that the fusiform bacillus and the accompanying spirochete are one and the same, the latter being an evolutionary form of the former but always present with the bacillus.

2. The author believes that Vincent's angina is much more common than is at present realized, and that it is not recognized because of the failure to take a smear, and that furthermore it is frequently mistaken for diphtheria and syphilitic ulcers.

3. It is also probable that the considerable use of candy or proteins with the subsequent lack of oral cleanliness predisposes markedly to this infection; likewise, that poor teeth harbor the organism and that it will manifest itself in its characteristic lesions whenever a suitable opportunity is given. It seems to be more frequent during the winter months.

4. The use of arsphenamin dissolved in glycerin is strongly advised in the treatment of Vincent's angina and infections in which the fusiform bacilli and spirochetes resembling Vincent's organism are found.

Emil Mayer.

IV.—LARYNX, TRACHEA AND ESOPHAGUS.

Thyrotomy and Laryngectomy Under Local Anesthesia in Cancer of the Larynx.

Moure, E. J.

Rev. de laryngol., etc., Paris, 1920—XLI—1.

Opening the larynx, according to Moure, no longer is a formidable operation. Postoperative bronchopneumonia is no longer to be apprehended. Thyrotomy is a desirable operation in cancer of the larynx when the disease is well limited to one side. Like laryngectomy, it is always to be performed under local anesthesia. Moure has performed in all, thirty-four laryngectomies, of which five died subsequent to the operation. In the last fifteen cases, only one death occurred. The good results he ascribes to improved technic, especially the use of local anesthesia, and the performance of a tracheotomy at least fifteen days before. He does not favor suturing the tracheal opening to the edges of the skin. The introduction of a large canula into the wound, reinforced by gauze, is sufficient. The placing of an esophageal sound and of the "Lombard" canula assists much in the subsequent dressings. Great emphasis is placed upon the care in after-dressings. In Moure's opinion, even if the tumor is limited to one side of the larynx, a total laryngectomy is to be preferred on the ground that it is not more mutilating than a hemilaryngectomy and gives much greater security. When the growth is extralaryn-

geal well localized on the free border of the epiglottis, it is possible to remove it by the transhyoid route, or better still, the transthyrohyoid. A diffused invasion of the esophagus with involvement of the glands renders the case inoperable. Harris.

Hemilaryngectomy.

Bremond.

Rev. de laryngol., etc., Paris, 1920—XLI—8.

The author is in agreement with others that theoretically hemilaryngectomy is to be reserved for the earliest and most localized form of cancer. He argues, however, that there are cases where a radical operation is indicated and would be refused, and where a less radical procedure might be accepted by the patient.

The operation should be done in two steps. First, the tracheotomy, and second, removal of the hemilarynx, the two steps of the operation to be separated by an interval of several days.

After reporting several operations, he concludes as follows:

1. Hemilaryngectomy is indicated in new growths of the larynx when they are well limited and when the patient refuses a total intervention.

2. The operation is much easier and less dangerous than the total laryngectomy.

3. The patient with his hemilarynx has the possibility of breathing normally and making himself heard. Harris.

V.—MISCELLANEOUS.

Adenoma of Mucous Glands of Mouth, and Macrocheilia.

Hatton, Edward H.

J. Am. M. Ass., Chicago, 1920—LXXV—1176.

Enlargements of the lips produced by disease of the mucous glands are uncommon, and are either inflammatory or neoplastic in character. Volkmann, in 1870, under the name of myxadenitis labialis, or cheilitis glandularis apostematosa, described five cases of a chronic inflammation of the lip mucous glands; all the patients were adults, three had syphilis, and the process was long drawn out with frequent exacerbations. These mucous glands were palpable, the gland ducts frequently dilated, and small cysts or abscesses common. Stomatitis or gingivitis was present to a noteworthy degree in every case, and Volkmann believed that the gland involvement was due to the mouth infection and that syphilis when present was a factor of only secondary importance. A more frequent enlargement is that described as "double lip" by the European writers, especially the French and English, sometimes called hypertrophy of the mucous or labial glands. The enlarged soft lip is covered by smooth mucous membrane, with palpable nodules beneath it. The fold formed by the projecting red portion is more conspicuous when the person is laughing or talking. In the upper lip the fold is divided by the frenum, and the lower is always single.

Of nineteen recorded cases, only one was in a female, thirteen involved the upper lip, nine the lower (this included three of both),

five of the lower lip class were in syphilitic individuals, and two were of supposed inflammatory origin. Two were associated with trauma, one patient had misplaced teeth, two had other deformities, and six had syphilis. The neoplastic form was found in the upper lip twelve times and the lower three, and began in childhood, if not earlier, almost without exception. There is no statement anywhere connecting these tumors with malignancy. Excision, if extended to include all of the involved glands of this form, has been uniformly successful.

The term microcheilia therefore has been used to name not only the better known lymphangioma but also these unrelated mucous gland enlargements. It is difficult, if not impossible, to classify these conditions on the basis of any such terminology, and it would therefore seem wise to abandon such descriptive terms as microcheilia and double lip for the more exact names associated with the actual pathologic condition that does exist. Emil Mayer.

Prevention of Respiratory Diseases.

Lynch, Charles.

Med. Rec., New York, 1920—LXCVIII—801.

Our conclusions from our first study were as follows:

1. Influenza, as well as other socalled airborne or respiratory diseases, is essentially a hand to mouth infection.
2. There are major and minor modes of transmission by indirect contact—by the former through tepid mess kit washwater, and by the latter through intermediate inanimate objects, the hand being the conveyor in both instances. Among troops approximately 75 per cent of the cases arise by the major route. This applies to troop transports as well as camps.
3. Transmission by direct contact is only subsidiary to the indirect routes.
4. The adoption of a universal method of washing mess kits in boiling water will enormously reduce the prevalence of all mouth borne infections.
5. The prevention of hand to mouth infections may be accomplished by any measure which will prevent the unclean hnad from visiting the mouth and nose, and by the thorough cleansing of the hand after all such visits.
6. The incubation period of influenza would appear to be from two to three days.
7. An epidemic of influenza among troops may be controlled by elimination of hand contaminated dishwater.

Emil Mayer.

A Clinical Study of Epithelioma of the Lower Lip.

Lain, Everett S.

J. Am. M. Ass., Chicago, 1920—LXXV—1055.

1. Considering the gravity of epithelioma of the lower lip and its frequency in adult males, we believe the subject has not been given the study and attention it deserves by American physicians.
2. As noted by others, our reports also show epithelioma of the

lower lip to be far more common in outdoor workers, and most frequently seen on the side of the mouth where a cigar or pipe is held.

3. Early diagnosis and treatment of the lymph drainage of the lips will materially raise the percentage of cures.

4. Neither surgery, radiotherapy nor any other one successful method of treatment should be used in all cases alike.

5. Radium and the roentgen ray, singly or combined, give the most satisfactory results in a selected class of epithelioma of the lower lip. Emil Mayer.

Tetany Following Thyroidectomy.

Poppens, Peter H.

J. Am. M. Ass., Chicago, 1920—LXXV—1069.

Owing to the fact that the tetany appeared and disappeared simultaneously with the infection, this was probably the principal etiologic factor. Thyroid extract apparently alleviated the acute symptoms and tided the patient over the critical period.

Emil Mayer.

Plastic Surgery: Its Interest to the Otolaryngologist.

Smith, Ferris.

J. Am. M. Ass., Chicago, 1920—LXXV—1555.

Three general methods cover all types of plastic operations on soft parts:

A. "French Method."—This consists of sliding or stretching of the flap with little twisting or torsion of the pedicle, and is widely used alone or combined with other types for repair in all parts of the body. The essential characteristic is masking a loss of substance. It should never be used in large parietal or total losses of the nose, because rapid flattening and deformity result. Its use in repair of a cavity always demands a lining of mucous membrane or skin.

B. Interpolation.—1. Flaps from the immediate neighborhood, either single or double, with a pedicle which is rotated, twisted, bridged or tunneled. This is the Indian mutilation type of flap. The pure Indian flap was raised from the forehead, rotated on a pedicle and used without lining for nasal repair. This [ap is the method of choice for nasal covering and the method of necessity in covering large losses of the cheek and lips.

2. Flaps from a distance with a tubed pedicle. Tubing of the pedicle is a valuable modification by Major Gillies. This consists in suturing the raw edges of a long pedicle, thus doing away with danger of infection by including the raw surface and furnishing a certain blood supply to a flap at its distal end. Perthe's method of outlining, dissecting and resuturing the flap in its original bed is usually combined with this procedure.

3. Free flaps or grafts. These may be skin, fat, fascia, bone or cartilage.

4. Flaps transferred on a "carrier," usually the arm. This is essentially the "Italian method," which was primarily proposed

for rhinoplasty. It consists in fashioning a pedicle on the arm and later transferring it to the nose with the arm immobilized on the head until new blood supply is established.

In the light of present knowledge, the method has nothing to recommend it in facial repair, and many things to condemn it. The position is torture to the patient, there is danger of emboli in the superficial veins of the arm, infection readily occurs from contact with the nose, and dressings are difficult. Its supporters can only claim that it prevents scarring of the forehead. This is offset by the fact that the texture of the skin is such that its contrast with the face and scalp is very marked.

If forehead skin is not available, chest skin should be supplied by use of Gillies' tubed pedicle.

C. Retrenchment.—This covers operations for reduction of excess tissue and production of a more pleasing cosmetic result.

Emil Mayer.

Society Proceedings.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday, March 1, 1920.

THE PRESIDENT, DR. ALFRED LEWY, IN THE CHAIR.

Paraffinoma.

Dr. Harry L. Pollock presented a patient who had been operated upon for paraffinoma, and exhibited photographs taken before and after the injection of the paraffin and following operation.

The patient was a young lady who had a slight "saddle nose," for which a beauty specialist had injected paraffin to remedy the defect. The paraffin soon produced irritation and a large growth resulted, which included the corner of her eyes and part of the forehead. She consulted a surgeon in Minneapolis, who decided to remove the growth, and immediately after this the nose took on the appearance of acne rosacea. The paraffin was injected eleven years ago and operation was attempted two years later.

When seen by Dr. Pollock two years ago, she presented an irregularly lobulated tumor mass, which caused severe pain in the eyes. For this reason removal was decided upon, so part of the mass was extirpated and the wound kept open. Evidently the air had some effect in dissolving the paraffinoma that remained. After a few months they dissected out the whole thing, so far as they could teli, but after a short time there was a recurrence that was almost like a malignant growth, which went down to the antrum and involved the tissues of the face. Following this they performed a radical antrum operation on both sides, making through and through drainage, and dissected up under the eyebrows on each side to remove the paraffinoma and permitted the wound to granulate in. Many different things were used to assist in healing, but until the institution of radiotherapy four weeks previously, there had not been much change. At the time of presentation the pain was all gone and the wound was healing over. After the wound was entirely healed they expected to do some plastic work, and would probably make a new portion of the nose from a piece of the tibia.

Dr. George E. Shambaugh presented a paper entitled

Discussion of an Old Problem.

Dr. Shambaugh introduced his discussion by relating several experiences that had come to him recently. The first was a case of typical otosclerosis, who had been subjected to a series of operations on the nose and throat, including removal of all four turbinated bodies, resection of the nasal septum, tonsillectomy, and

curettement of the nasopharynx, all undertaken with the mistaken idea that the patient was suffering from a form of ear trouble which might be caused by nose and throat conditions. The second was a case sent to him to have all these operations in order to prevent a loss of hearing. The patient was suffering with tinnitus, probably due to the initial stages of otosclerosis. There was no middle ear disease. The third was a patient who was being urged to have a radical mastoid operation on the one ear on which she had to rely for hearing. The examination disclosed a harmless type of chronic ototis media, with a perforation in the anterior segment of the membrana tympani and a slight mucous discharge: an ear condition quite free from any risk of a serious complication. The fourth was a patient who for a couple of months had been suffering from an acute otitis media, with palpable evidence of a softening within the mastoid. This patient had been urged to have the turbinated bodies removed, the septum resected and the tonsils removed, in order to correct the otitis media.

All of these cases presented the most elementary clinical problems which confront the otolaryngologist, and in each instance entirely wrong advice had been given.

Dr. Shambaugh pointed out that this state of affairs is the result of the practice of teaching general practitioners the technic of the operations in the nose and throat, without teaching them the fundamental principles on which the practice of otolaryngology is based. It is a relatively simple matter to instruct students in the technic of operation, but it is a much more difficult matter to teach them to make the proper examination and the diagnosis of the conditions requiring these operations. It is not surprising that the wrong advice is so frequently given or that so many unnecessary operations are being performed.

Dr. Shambaugh insisted that the remedy must be a reversion of the practice of teaching operations to men before they have been taught the principles of otolaryngology and the methods of diagnosing the condition requiring these operations. He insisted that the minimum time to be spent in preparation for the practice of otolaryngology should be one year full time work, in which as much time should be spent in the study of the fundamental sciences of anatomy, embryology, physiology, etc., as in the clinical aspect. The technic of operation should be taught only as the final part of this preparation. The need is not so much for a large number of specialists in this field as it is for a few properly trained men to do this work. We owe it to the public, as well as to our specialty, that we take a definite stand on the minimum requirement of preparation for practice in this field. This is more necessary now than it was some years ago, when it was the custom of general practitioners to treat these cases by means of sprays and topical applications. The teaching of these men the technic of an operation has thrown open the gates for immeasurable harm from unnecessary operating.

Dr. Shambaugh pointed out that this was not a new problem but that in 1907 ("The Preparation of the Specialist," Jour. Am. Med. Assn., 1907, XLIX, p. 540-543) he had written on "The Preparation of the Specialist," in which he had insisted that this work should be undertaken by the university, and that the work should be put

on the basis of real graduate work leading to the granting by the university of the higher degree, the degree of Doctor of Philosophy, for example, in otology. That in 1909 he had discussed the same subject in a paper entitled "Postgraduate Instruction in Otolaryngology," which was published in the Laryngoscope ("Postgraduate Instruction in Otolaryngology," Laryngoscope, June, 1909). Again, in 1912, he had discussed "The Specialist in Medicine" ("The Specialist in Medicine," Jour. Amer. Med. Assn., LXIII, p. 1827-29), in which he stated that the real specialist in any subject was one who mastered the subject sufficiently to attack the unsolved problems in that subject. That the specialties were no place for the dilettante or the unsuccessful general practitioner. He also pointed out the analogy between the work of the postgraduate schools in the training of specialists and that of existing preparatory medical schools, and insisted that the proper place for both was in the properly equipped department of medicine of the university. He pointed out that since these papers were written the undergraduate institution in medicine had been taken over by the university, and he believed that the time has come for the passing of the postgraduate school as an institution for training men for practice in the medical specialties. When this work is taken over by the university there will be less effort made to ladle out to the student medical facts in courses devised for this purpose. Instead, the effort will be made to teach these men how to investigate the facts. The chief reason why, from all the men who in the past have crowded our postgraduate schools and filled the courses offered abroad, so few have ever been contributors to the advancement of our specialty, is because these courses have aimed to teach the student the established facts in the specialty, rather than to train him to investigate these facts.

DISCUSSION.

Dr. Norval H. Pierce hesitated to open the discussion because Dr. Shambaugh was quite conversant with his views on the subject. The frequency with which the profession was brought in contact with cases that showed the crassest kind of ignorance regarding diseases of the ear, nose and throat, especially of the ear, was pitiable and a matter of considerable importance. The war had disclosed the fact that a large percentage of men who claimed to be fitted for entrance in the otolaryngologic section of the Army were ignorant of the elements that go to make up a specialist in that branch. The cause of this was two-fold: first, the lack of facilities for training otolaryngologists in this country and abroad and, second, the commercial spirit of the age. If a man could learn the technic of tonsillectomies, he cared very little about the diagnosis of cases that would give him some idea regarding the advisability of tonsillectomy. If he could learn the technic of any operation, the fact of whether or not it was necessary to do that operation was pretty far back in his mind. The man with a family and no other means of supporting them than that which he got from the practice of medicine was a very human individual. It was easy in certain cases for the fee to turn the judgment in the direction of the operation—there was no doubt about that.

Therefore, training in this specialty not only enlightened a man but built up his character. A man who really had as much knowledge as could be obtained in these matters was a good deal better man as regards his medical character than the man who had only a smattering. He did not blame the men especially; the foundation of the trouble was ignorance, not dishonesty.

Dr. Pierce believed with Dr. Shambaugh that the time was rapidly approaching when facilities would be extended to men to fit themselves for a beginning in the pratcice of otolaryngology. One year of intense study was the very shortest time that should be devoted to this preparation. He believed they should begin with the teaching of anatomy and physiology, and with the physics of the specialty, if necessary. Graduates should not be admitted to the latter part of the course, which comprised clinical work, diagnosis and operations, until they had had preparation in the fundamentals. In fact, he thought it would be well for men in different parts of the country to get together and make certain requirements in this line. There might be an exchange of privileges—a man could take the first three or six months in one university and complete the course in some other university, but the whole thing had to be formulated. The fact that the postgraduate course of six weeks was being abandoned in many directions was a sure sign that it was about to pass away.

Dr. Otto T. Freer had not found that the many physicians who have entered into otolaryngology insufficiently prepared have done so from mercenary motives. He thought they merely had no conception of what is asked in effort and fundamental knowledge to make an otolaryngologist. The fault is less with the men than with the poor opportunities for study offered them. Impoverished graduate schools, with insufficient equipment and unpaid teachers, who can give to the school only the time they rob from practice, cannot produce specialists of merit. Reform will not be had until the public appreciates the importance to itself of the best possible educational facilities for the doctor and stops blaming him for its own neglect to supply them. What is needed above all things for him is an unlimited supply of fresh or properly preserved (not formalinized into leather) cadavers for continued, lifelong study of the topographic anatomy and surgery of his chosen specialty. Without such abundance of cadaver material graduate teaching, no matter how much exalted with university formalities and capped and gowned, will lack the power to give the student diagnostic and surgical confidence. Vienna became the greatest graduate school in the world largely because of the immense cadaver material offered there, and because the national government gave the teachers an authority that made the public obedient and respectful to them. The strengthening effect of government backing upon the profession is evident in Michigan, where the general public looks up to the graduates of the University of Michigan with a deference not shown by other States towards their doctors. State or Federal medical schools to replace privates ones would do much to change the attitude of the public towards the profession from one of impertinent doubt into confidence and respect.

Dr. Eugene R. Lewis (Dubuque, Iowa) said he thought it was a fruitful subject for discussion and thought, although it was noth-

ing very new. The same problem had always been present in one form or another. The time was when, if a boy decided to go into medicine, he allied himself with a man who was already in medicine, and after a time he was in medicine, too. These conditions were not satisfactory, and finally certain requirements were stipulated for the degree of M. D. Then arose abuses, the M. D. "diploma mills."

He had heard one of the older triologic members tell how he became a specialist, how very crude his entrance was into the specialty, and yet there was then no other avenue of entrance. He thought another stage in development had now been reached, following a natural course of evolution. The problem was not easy to solve. He asked Dr. Shambaugh if the idea that he had in mind was that a man should go into the study of otolaryngology without an underlying foundation of years of practice. It was a very difficult problem to decide just how much general work a man should have before taking up special work. Everyone had seen the unfortunate narrowness of the man who was "only a specialist," he who had entered his medical school with the intention of becoming a specialist, and had gone through with only perfunctory compliance with the required subjects in the curriculum, but intent only upon his specialty. He thought those men were not capable for the highest type of special practice; for this, he believed, it was desirable that a man should have a university degree, a medical degree, an internship in a good general hospital, and then the practice of general medicine for a certain length of time before becoming a student of otolaryngology. This was a big requirement, but the man who did not do it could not be the highest type of otolaryngologist, for a background of general medicine was necessary for the highest type of any specialty. The profession is not up against a new problem at all, and the present generation would probably see the solution of the problem.

Dr. George W. Boot thought a slightly different view should be taken, which was that if the undergraduates were better instructed in ear, nose and throat work they would demand more of the specialist when they got out of school. The better prepared the student was in medicine, the better prepared the otolaryngologist would have to be to get his work later. If sufficiently well founded in otolaryngology, there would be no danger of his sending his work to a poorly equipped specialist, for he would know a well equipped specialist when he saw one.

Dr. J. Holinger thought that in the training of specialists they ought to take advantage of the opportunities at their disposal at the Cook County Hospital, where there is very good material. When he started work there he made it a point to have a resident assistant for at least a year. The assurance was given him by the Civil Service Board, but after three months the assistant was changed. After another three months there was another change. Such conditions make it impossible to train specialists.

Dr. Holinger agreed with Dr. Shambaugh that many unnecessary operations are done, but sometimes, too, the patient is to blame for this. He cited the case of otosclerosis combined with occlusion of the tubes in a heavy smoker with a pronounced hypertrophy of the lower turbinates. This hypertrophy was removed, which gave much

relief to the patient. Three years later the man was again having trouble and someone told him the tonsils should be removed. This was done and again the patient felt better. Owing to incessant smoking, he got into trouble again, and at a third place had another operation performed. It is not always the doctor's fault if unnecessary operations are performed, for they do not always get the truth from the patients. On the other hand, they were forced to admit that they sometimes got results where they did not expect them. An old lady of over seventy years suffered from hard hearing and dizziness, and obstruction of the nose. She had hypertrophic lower turbinals. Dr. Holinger thought that most of the symptoms were due to old age, but on repeated insistence of the patient that something be done for her nose, he removed the free ends of the turbinals. The result was unexpectedly gratifying. Besides a perfectly free nose, all the ear symptoms disappeared.

Dr. Shambaugh (closing) said that where nasal conditions existed which in themselves required correction, they should, of course, be taken care of, but what he wished to emphasize was that the existence of some irregularities of the nasal septum or the fact that the patient has a nose at all, or tonsils, seemed to be sufficient in the minds of many to associate these conditions with ear trouble and to justify operations upon the septum and tonsils.

The question of internes operating upon the nose or throat was a very important one. They were all anxious to do everything they could to develop internes while they were in the hospital. The question of teaching them a technical operation like the septum operation or the tonsil operation hinged on this point: Had they a right to teach people to operate who were not prepared to diagnose the conditions for which they were operating? The decision as to whether tonsils should be operated upon hinged upon a number of important relations. The decision in many cases could not be reached without a postnasal examination. No interne that he had ever seen had ever acquired ability to make a postnasal examination. The decision to operate upon tonsils and adenoids was often made because of conditions found from an otoscopic examination. Dr. Shambaugh did not believe any interne ever acquired enough skill in making an otoscopic examination to determine the existence of those conditions which indicate adenoid and tonsil trouble. He believed with Dr. Holinger that these operations should be in the hands of resident specialists, and could see nothing but harm resulting from training a general practitioner in the technic of a nose or a throat operation.

As to Dr. Lewis' question regarding how much experience in general practice a man should have before taking up a specialty, there was no denying the fact that if a man practiced general medicine for five or ten years he was in a sense better qualified to practice in any special field than he would be if he went directly into this line of work from his hospital interneship. In the same way, a man would be a better laryngologist and otologist if he had five or ten years spent in neurology, etc. It was only another way of stating that a man would be a better specialist if he knew everything there was to know about medicine; but it was evident that this was not possible and that they must have some rational basis upon which to work. In Dr. Shambaugh's opinion, a man who

took up a specialty should be a graduate from a first-class medical school and should have an interneship in a general hospital; but he questioned the advisability of expecting these men to spend five or more years in general practice before starting on their special work, which meant, as a rule, from two to three years' preparation.

The solution of this problem would be by cooperation. This was the great advantage of specialization. They did not look with approval upon a general practitioner who attempted to tinker with the specialties. In the same way, the internist had as much justification for disapproving of the specialist attempting to practice general medicine. Their best results were obtained by working in cooperation with men who were specializing in the various lines of medicine. For example, the decision as to whether tonsils should be removed was not one that could be decided in all cases by a man doing the nose and throat work. In many cases the decision hinged upon whether the patient was suffering from a systemic infection and also upon whether there was any other likely focus which might account for the systemic symptoms. In other words, the decision to operate was very often better made by the internist than by the throat specialist.

Dr. Samuel Salinger read a paper on

Primary Periostitis of the Mastoid.*

*See page 943.

DISCUSSION.

Dr. Otto T. Freer thought Dr. Salinger's paper a very good presentation of the subject of external abscess of the mastoid process. The disease was rare and hard to recognize. In the diagnosis the most important thing was to see the drumhead. Here an ear speculum of the right shape was necessary. Most specula had a conical shape and could therefore not be passed through the external auditory meatus when narrowed by elastic inflammatory swelling, as the yielding of the swollen skin formed an excellent cushion for the cone of such a funnel to wedge itself into, the more one pushed the more it wedged. The ear speculum that suited such a state should have parallel walls and have a caliber of only one-eighth of an inch. With the aid of such a speculum, Dr. Freer had been able to see the membrana tympani, where an ordinary funnel would not penetrate. A neecssary accompaniment of inspection with so minute a speculum was, of course, penetrating illumination, which was not obtainable with a head mirror and only to be gotten with the Kirstein lamp. It was unfortunate that at present no Kirstein lamps were obtainable, but Sharp and Smith said that they would have Kirstein headlamps in the market by July 1st at the farthest. In the interest of bronchoscopy and other deep illumination this is highly desirable.

Dr. Eugene R. Lewis (Dubuque, Iowa) stated that he had a very interesting case in December, following a septal resection in a boy of seventeen, who was in very poor physical condition, and the resection was followed by an acute otitis media. Between Christmas and New Year's he attended some party. The next day the ear discharge ceased abruptly, and within twenty-four hours he had

a tremendous swelling of the mastoid, apparently a mastoid abscess. The boy had quite a lot of pain, and it was a typical looking case, with a swelling of the posterior wall. The patient was kept in bed and observed under tentative treatment for five days, but the only result was further dissemination of the swelling. X-ray plates showed intercellular partitions rather clearly. He told the father of the boy that it was a somewhat atypical case and he did not think it was a mastoid abscess, although it was suggestive of that condition. He had enlarged the perforations the first day, but could not get any discharge except a little mucopurulent material which soon ceased. He suggested making a posterior opening for inspection; if it was a periostitis they could save the mastoid, and if not, simple mastoid drainage could be done. This was done under local anesthesia. They denuded the periosteum very widely, looking for periosteal pus or signs of cortical change, which they did not find. They went into the neck for two or three inches and opened up as wide as they could, so as to get free drainage. The wound closed in three weeks, and there was no discharge from the middle ear after the fifth day. He did not know what the cause of the trouble was, but evidently acute extension of the infection caused some interference with the circulation.

Dr. Salinger (closing) said there were some points in connection with these swellings that could be cleared up. He thought there was a field for investigation in reference to the lymphatic supply, which he believed had something to do with the swellings. He did not know why these things would puff up, and when incised, promptly subside. This had been reported a number of times in cases of enormous swellings with pain and edema that recovered after being simply incised and sewed up, but the etiology was not known. The point he wished to make was that with a swelling over the mastoid and a swelling above the meatus, it was only natural to suspect a mastoid infection; yet without a history of previous discharge one might be at a loss. He had not been able to push aside the swelling in the meatus with a funnel (as suggested by Dr. Freer) because of the great pain. He knew that Dr. Freer was a great enthusiast about the Kirstein light and agreed that it was a valuable adjunct in the examinations.

THE CHICAGO LARYNGOLOGICAL AND OTO-

Meeting of April 5, 1920.

THE PRESIDENT, DR. ALFRED LEWY, IN THE CHAIR.

Otitic Meningitis.

Dr. J. Holinger presented a nurse who had suffered from suppurative otitis with swelling and tenderness over the mastoid and brain symptoms for several weeks. She heard only loud conversation in the right ear. After the mastoid was opened improvement for some time was noticed, but all symptoms, especially the unbearable backache, became worse. Lumbar puncture showed fluid under increased pressure, turbid and full of streptococci. After an improvement of several days, all symptoms reappeared, and especially the backache was severe. The wound looked well. With the intention of draining some of the cerebrospinal fluid, and at the same time looking for a possible brain abscess, the wound was enlarged and the dura over the cerebellum and over the temporal lobe were incised. Several stabs in the cerebellum and the temporal lobe showed no pus. The result was temporary. To relieve the backache, ¼ gr. of morphin was ordered every two hours by the interne without result. It was discontinued and 15 grs. of aspirin every evening seemed to have a very beneficial influence. The unusual features of the case were:

1. In the presence of symptoms of meningitis like slow cerebration, Babinski's and Kernig's symptoms and impossibility of approaching the chin to the sternum, the patient was able without pain to turn the head from side to side. Headache and sleeplessness were absent during the whole duration.
2. The diagnosis of meningitis was made certain by the finding of turbid cerebrospinal fluid under high pressure, containing many streptococci. This usually makes the prognosis fatal.
3. The decided turn for the better came with the medication of aspirin.

Cholesteatoma.

The second patient was a man of thirty-six, who came on account of loss of hearing in his right ear since last summer. He was treated in 1906 by Dr. Hardie for humming in the ear. After removal of adenoids and a few inflations he was cured in the summer of 1919. He never had humming of ears or pain. At the end of January, 1920, Dr. Holinger found on the roof of the external canal crusts which were removed with difficulty. A large marginal perforation in the drum membrane was seen and some granulations on the roof of the canal. In the course of about six weeks two pieces of cholesteatomatous masses, the size of a pea, forced their way through the perforation in the canal and through the granulations. At the time of presentation a third piece, a white mass between the red crown of granulations, was working its way into the external canal.

DISCUSSION.

Dr. Robert Sonnenschein thought the first case was probably one of streptococcic meningitis. That these may recover at times was shown in a case reported by Dr. Weaver about a year ago. Dr. Sonnenschein saw this patient during Dr. Friedberg's absence on military duty. The patient was a nurse who contracted the disease while on duty in the hospital. All the typical signs of meningitis were present, but there was no involvement of the nose, ear or throat. Several spinal punctures were made and the antistreptococcic serum injected. The patient made a complete recovery after three or four weeks.

Dr. Harry Kahn said he had seen the side to side movement of the head in a case of meningitis under his care.

Dr. Norval Pierce said it was very unusual for a case of meningitis to recover when viable microbes were found in the cerebrospinal fluid. None of his cases had recovered, and he wished to know whether the microorganisms were viable. Dead organisms could be recovered from a localized meningitis when the prognosis was less unfavorable, but when the microorganisms were viable it made the prognosis very grave. The fact that there was no headache was difficult to explain, especially as the spinal fluid seemed to be under pressure. Headaches were caused by increase in the cerebrospinal fluid and not by inflammations per se. Another colleague had a case of meningitis that had been going on for some time, from which the pneumococcus had been recovered. The patient had not yet recovered, but showed improvement. (The patient has since died.) Dr. Pierce thought one should be careful in giving even a hopeful prognosis in a case of meningitis where viable organisms had been recovered. It must also be remembered that accdiental contamination occurred in these cases from the skin, and therefore the technic in the spinal puncture and examination of the fluid should be very exact and very thorough. Much deepnded upon the fact as to whether viable microorganisms were recovered. In his opinion Dr. Holinger's case was not a general, spreading, suppurative meningitis, but in all probability a localized meningitis. How the viable microorganisms got down into the lower portion of the spinal canal it was difficult to say.

Dr. S. A. Friedberg thought that much depended upon the virulence of the microorganisms. It is well known in sinus thrombosis for instance, that the finding of hemolytic streptococci in the blood cultures, does not necessarily imply an unfavorable prognosis. Several cases that had come under his observation have recovered. Virulence tests on guinea pigs were negative. By analogy, it is possible that we may also have bacilli of low virulence in the spinal fluid.

Dr. Holinger (closing) stated that the cerebrospinal fluid was reported to be swarming with streptoccci, not one here and there as was often the case. The pathologic findings in the laboratories of the Cook County Hospital are pretty reliable.

Dr. Holinger was glad to hear that the peculiarity of free movement of the head from side to side in presence of absolute impossibility of movement of the head forward, was noticed elsewhere,

as Dr. Kahn mentioned. He would like to know whether this is in connection with the favorable termination of the case, in other words, whether this peculiarity is of prognostic value.

Dr. Stanton A. Friedberg read a paper on

The Carrier as a Factor in the Control of Diphtheria

DISCUSSION.

Dr. W. W. Hamburger (by invitation) said that he was quite at a loss to discuss Dr. Friedberg's paper on diphtheria, because his experience was confined almost entirely to the streptococcus. During his military service he worked during a period of eleven months on the streptococcus carriers, particularly in measles. They had isolated the carriers from the noncarriers and studied the effect on the carriers throughout the camp. In the early winter of 1917-18, when the measles came into Camp Taylor, all the men were very carefully examined. The measles cases entering the Base Hospital through the receiving ward were swabbed by the nose and throat surgeon and examined for streptococci, blood agar plates made, and the cases labeled either as carriers or noncarriers. At the end of twenty-four hours they were sent either to the measles carrier ward or to the noncarrier ward, so at the end of every twenty-four hours the receiving ward was emptied for new cases coming in that day. During the period in the observation ward the cases were carefully cubicled, the attendants carefully masked, and all measures of quarantine were used that could be carried out. The study resolved itself into a study of the complications arising in the carrier and noncarrier wards. The cases coming in during the height of the epidemic showed that seventy-seven out of one hundred carried streptococcus in the pharynx. As to complications, about 30 per cent developed in the 500 cases so studied. Of this 30 per cent, 6 per cent developed in the noncarrier wards, and 24 per cent in the carrier wards. The complications were the usual complications of measles, otitis media, acute sinusitis, acute bronchopneumonia, severe bronchitis, erysipelas in some cases, etc. The study covered a period of about two months, and they concluded that the segregation of measles patients into carrier and noncarrier wards was advantageous, resulting in a decrease of complications, for they proved that it cut down the percentage from 30 per cent in the carrier wards to 6 per cent in the noncarrier wards.

Later experiments showed that some of the complications were mixed infections. Sometimes they recovered pure streptococcus, sometimes pneumococcus, and sometimes mixed cultures. While it was found that complications could be diminished by carrier segregation the quarantine with masks and cubicles was not perfect. A survey was made every week and showed from time to time the development of carriers in noncarrier wards, and it was very often very difficult to determine the source of the carriers. Several times they found it occurred in attendants on the wards. Nurses or orderlies were found to be carriers and then were promptly excluded.

This work was carried further by immediately swabbing the new recruits as they came off the train into camp. Upon entrance into the camp about 15 per cent were shown to be carriers. At the end of six months this had been increased to 83 per cent, showing that the carrier state, at least in this study, had developed from contact with their fellows, and that finally the camp became heavily seeded with streptococcus. Another interesting thing was the variation in carrier per cent in epidemic and nonepidemic periods. In nonepidemic periods only 8 per cent were carriers, while 35 per cent was found during the period of epidemics.

During the height of the epidemic Dr. Hamburger visited Camp Grant and saw Dr. Capps and his method of masking which was in use there, and as soon as he got back to Camp Taylor he instituted the same method. While he felt that a step in advance had been made, the masking was not 100 per cent efficient.

To decide on the efficacy of the mask and cubicle method, they took a ward and planted a bed with a carrier and the next bed with a noncarrier and so on. In that ward it was absolutely impossible, by the most careful measures, to maintain that carrier separation, for at the end of one week, there were only three noncarriers left out of an initial number of twenty-five. So it seemed that the only way a separate noncarrier study could be made was that the ward itself should be maintained as a clean ward throughout, with all the unclean or carrier cases put into carrier wards.

As to the question of the meningococcus carrier, the feeling at Great Lakes was that this carrier was not of importance in the meningitis, while at Camp Taylor they searched for these carriers and kept down their meningitis pretty well, still they did not think that the carrier was much of a factor in the spread of meningitis. There were so many other conditions that entered in, that the simple finding of a carrier or noncarrier would not decide the question.

Another interesting angle of their work which checked up with Dr. Friedberg's was the question of how long the carrier state is maintained after an acute illness. They swabbed all the convalescent measles cases on discharge from the hospital, finding about 72 per cent carriers after recovery from their measles, in spite of all local treatment. In the laboratory they tried out various antiseptics in various strengths, and various combinations to see what was a useful antiseptic to use for killing the streptococcus, and found a 2 per cent iodoglycerin in vitro was the most valuable; but this proved absolutely useless as a gargle. A certain group of the 72 per cent convalescent carriers were put out on porches, with abundant fresh air and sunshine and ample food and some exercise, and within forty-eight hours the carriers dropped from 72 to 40 per cent. Following this they gave up all local measures and kept the patients for a week under conditions of fresh air, sunshine and good food, and the percentage of carriers dropped promptly.

Tests had demonstrated that in the parts of the camp where measles were prevalent the dust of the barracks showed a high

percentage of streptococcus, while in the carrier wards the percentage was very low.

Capt. Kleinman (Fort Sheridan, Ill.) said that he had charge of a group of diphtheria carriers in November. Then cases in all were cultured, both nose and throat, for the first time, and out of that group two were found to be ethmoid carriers, and they had been longest in the hospital. The study was continued until after the first of the year and out of the seventeen cases about 35 per cent were found to be ethmoid carriers. Since that time all of the cases have been cultured, both nose and throat, and the treatment with argyrol tampons, as outlined by Hazeltine, has been carried out. In this way the time lost in hospital had been cut down. The ethmoid group averaged thirty-three days and the nonethmoid about thirteen days. The treatment consisted of quarantine, masking when out of their ward, applications to the nose and throat, and gargles. Cultures were made Mondays, Wednesdays and Fridays, and three consecutive negatives from both throat and ethmoid region were required for release. Tonsillectomy had not been done.

Major W. F. Von Zelinski thought the carrier question was a collosal one and could be discussed indefinitely. He had seen a good deal of it in regard to measles, meningitis and diphtheria, but had been unable to form any conclusions. He had listened to a very exhaustive paper by Dr. Davis at a recent meeting of the Society and left with the conclusion that everybody had organisms in their throat all the time of one kind or another. The virulency of these was a big question and one he did not wish to discuss extensively. The question of testing them out for virulency was a question of testing out immunity; on one person organisms might be very virulent and in another person not so virulent. They seemingly became more virulent by passing through a number of people, and also seem to lose their virulency in time.

He had hoped to hear something said about the treatment of carriers. During the last few weeks at Fort Sheridan the carrier patients (diphtheria) had been confined under treatment for thirty to thirty-three days. Recently no local treatment of any sort had been used, for they had found that on a simple gargle they became free from organisms as quickly as with the local applications used before. They had experimented with injection of the Schick serum and found that some of the men who had been free of the organisms for a number of days gave a positive reaction, and some who still had them gave a negative.

Major Von Zelinski was stationed at Camp Wheeler, Ga., from December, 1917, until April, 1918, and in the epidemic of meningitis in that camp there were no active cases in his regiment. The central inspectors came over to his infirmary with the dichloramin T spray and he was instructed to spray certain men. They took some cultures at different places, and if a carrier was found they swabbed all the men in that tent and then went into the next, gradually widening the circle as carriers were found. This was ineffective treatment, because it was impossible to reach all the places where the germs might locate. At that time the troops were receiving instruction in the use of gas masks, and they allowed

the men to pass through benzolchloride for about thirty seconds. Major Von Zelinski thought this might be an effective antiseptic that must certainly reach all the areas in the nose and throat, and that it might be a good idea to pass all the men through the gas house. The Chief Surgeon was against it, but he put the matter up to the commanding officer, who was enthusiastic about it. A bunch of men were taken up and swabbed before they went in and swabbed when they came out, and it was found that after this experience they were free of everything except the micrococcus catarrhalis. They then used this treatment on the carriers and found it reduced the period of isolation for fifteen days. While in Europe he saw a statement in a French medical journal that the Germans had been using this treatment, and ascribed the value of the effect to the fact that the treatment practically flushed all the mucous membranes. From the standpoint of treatment this was the most effective thing he had found. The bacteriologist said the meningococcus was pretty well kept away by this treatment, and one or two treatments was all that was required. They tried it on the pneumococcus and they remained away for two days. He mentioned this simply for what it was worth. The question was much too big for him. Theoretically, if all the laws of isolation were rigidly enforced it would be necessary to lock up everyone, for everyone carried something all the time, and just how dangerous these microorganisms were was yet to be discovered.

Dr. S. A. Friedberg (closing) said that an underlying pathologic condition must be present in order to establish or maintain the carrier condition. In the presence of epidemics in organizations or closely associated groups, the bacilli may also be found in normal conditions of the nose and throat; however, their presence is usually of a transient nature and they are easily gotten rid of.

In answer to the question as to immunity to diphtheria in children after the removal of tonsils and adenoids, the speaker did not believe any was conferred. The susceptibility to infection, in the absence of suitable lodging places for the growth of the bacilli, was materially lessened.

Dr. Hamburger had mentioned that they had taken air cultures in order to determine various sources of dissemination. In Dr. Friedberg's work, the cultures were made in every possible manner, and from every source including air, gas masks, kitchen utensils, etc. From the results, it was impossible to draw any definite conclusions. Very evident factors in the spread of disease were found in the main mess halls, large out-patient departments, and carelessness or ignorance as to necessary precautions to be carried out.

From what one of the speakers has said, there seemed to be a little uncertainty in regard to virulency and avirulency of the microorganisms. It is a well established fact that a virulent bacillus does not lose its virulency, nor is it possible for an avirulent bacillus to become virulent. The virulency of the bacillus is determined by guinea pig innoculation and not by the fact that a person may contract a severe clinical case by having been exposed to a patient only slightly ill from the disease.

Dr. J. Holinger read a paper on

Tonsillectomy in the Presence of a Peritonsillar Abscess

ABSTRACT.

The removal of the tonsils in the presence of a peritonsillar abscess is a procedure which as yet has undeservingly not many friends. Still it is certainly a logical operation, as the tonsils are the center of the trouble, and sooner or later have to come out, because peritonsillar abscesses often recur. The objections are more in the nature of prejudice which is not borne out by experience. The recovery after the removal is rapid. It does not take any longer than after an ordinary tonsillectomy.

The operation is more difficult than an ordinary simple tonsillectomy and the general anesthesia has to be given by an experienced anesthetist. The hemorrhage is usually more severe, but can be controlled by the usual methods. The anatomic conditions are more complicated on account of displacements by the abscess which may develop above or behind or directly lateral of the tonsil, or in the tonsil itself, thus dividing the tonsil in several pieces. The method of removal is by means of forceps, sickle-knife and snare. Septic symptoms which are often present in cases of peritonsillar abscess usually disappear in the first twenty-four hours, and albumen in the urine within the first two or three days.

DISCUSSION.

Dr. Louis Ostrom (Rock Island) said that eight years ago he started work of this kind and he had never seen a case that had not had just as good, if not a better, convalescence as a normal tonsillectomy. In the A. E. F., at Coblenz, it was put up to him to assume the responsibility. He was told by some that it was a criminal procedure, but every patient made a wonderful recovery (perhaps good luck), while at the same time all kinds of complications followed tonsillectomies in normal cases (perhaps bad luck). If the patient had double quinsy he operated just the same. His experience with anesthesia had been entirely local (one-tenth to one-fifth per cent. of cocain with adrenalin) except in children. He had had no trouble with this and saw no reason why one could not obtain perfect anesthesia. From the time the quinsy first appeared to periods ranging from a few weeks to several months he had had no difficulty in securing complete anesthesia. He never urged or tried to persuade a patient to have the operation done, but after explaining the matter thoroughly most patients demand it. In many cases the pillars had been a solid mass of scar tissue and the abscess had burrowed outside the constrictor muscles, exposing the large vessels. In only one of those cases he had some hemorrhage that was troublesome. In this case he had used a piece of gauze, taking a stitch on the side of the posterior pharynx and away forward on the side of the jaw to hold it in place. In this instance the result was good. The records at the eye, ear, nose and throat hospital, at Coblenz, will show that the novocain collapse was just as frequent as from cocain. He made the first injection to the side

of the uvula, going up and trying to follow up to the hard palate, down to the base of the anterior pillar, then behind, and infiltrating between these three points. In the paratonsillar abscess the constrictor is nearly always laid bare by the abscess itself. No anesthesia is needed through the capsule at this locality. In no instance had any of his patients complained of pain. The cases thus operated would probably reach two hundred or two hundred and fifty, and in all the anesthesia was entirely successful, even when done on both sides, when the swelling had almost entirely closed the throat. Using a large quantity of the anesthetic had no bad effect so far as healing was concerned. He was guided entirely by the sensations of the patient; if they felt the work on the tonsil, he used more anesthetic. If he expected to use a large quantity of anesthetic he would use even a weaker solution and depend on infiltration of the surrounding tissue.

Major W. F. von Zelinski said that in one case like this which had been operated under local anesthesia, in which there was nothing particularly difficult about the anesthesia, the patient afterward developed pneumonia, which he attributed to the aspiration of some pus. He had had two cases, and thought there were two points to consider: one, the hemorrhage was very severe, and the other was the danger of pneumonia. He did not care to take out any more tonsils when there was any paratonsillar abscess, but thought it much simpler to drain the abscess first and believed the chance for complications was less when this was done. He thought even Dr. Holinger would not deny that he was taking a chance of having some of the pus escape and set up a secondary infection in the lung.

Dr. Norval H. Pierce thought that this was such an unsurgical proposition that he must say something on the subject. Why a complete local anesthesia should be obtained in this region in the presence of inflammatory action when one was unable to obtain anesthesia in other parts of the body under the same conditions, was very hard to understand. He had some difficulty in producing local anesthesia even in cases where the paratonsillar abscess had been evacuated and some time had elapsed, because of the scar tissue that is produced. The excision of such tonsils is likely to be painful, although he thought he injected as deeply as the previous speakers. Surely one could not get local anesthesia to any great degree in inflammatory conditions, as was proven in the local anesthesia of the mastoid. One might as well not use any anesthesia where infiltration of the mastoid region had occurred or where there was a subperiosteal abscess. Dr. Pierce could see no advantage in this method of operating, and Dr. Holinger, in his paper, provided enough argument against it to discredit the operation. Dr. Holinger had stated that he was limited to a general anesthetic, which in this condition is the most dangerous anesthetic; he stated that the pus gushed out from various pockets and he believed this was a great menace under general anesthesia, inasmuch as it was likely to get down into the bronchial tubes and produce pneumonia. He said the hemorrhage was much greater, and this again was against the operation; his operation was inexact, inasmuch as he discovered

that he had not completely removed the tonsillar tissue; these facts were enough to discredit the operation. It was more dangerous, more difficult, less complete and nothing was gained by it. Dr. Pierce thought it was better practice to evacuate the abscess, which was not difficult, and allow a certain time to intervene before performing the tonsillectomy, when it could be done under local anesthesia without the dangers incurred in this procedure.

Dr. T. W. Lewis said that he had taken out the tonsil in the presence of an abscess repeatedly, but did not do it as a routine. He had stumbled on the removal of the tonsil in the presence of an abscess inadvertently; in removing a tonsil he had discovered an abscess which he had no suspicion of before operation, and that led him to think of the possibility of the removal of the tonsil in the presence of an abscess. Subsequently he had operated to evacuate an abscess and found before he discovered the abscess that the tonsil was almost completely enucleated. He then went on and took out the tonsil, but did not remove the second tonsil at that sitting. Subsequently in dissecting the tonsil to evacuate an abscess he found that the abscess was so low and external to the tonsil that the tonsil was again almost completely enucleated before the abscess was evacuated. Dr. Lewis was rather inclined to agree with Dr. Pierce that the operation was not in accord with strict surgical principles.

As for the anesthesia, it seemed to him that in the presence of an abscess in this location a general anesthetic was absolutely contraindicated. He would not think of giving a patient a general anesthetic and evacuating any large quantity of pus into the air passages. Sometimes the pus was under such pressure and was present in such large quantity that the whole throat filled at once and under a general anesthetic some of the pus was almost certain to be aspirated. It was not always possible to have an aspirator in position at the time the pus began to flow or to remove it at the desired time. He was still in doubt as to whether it was advisable in all cases to remove the tonsil under such conditions. In operating on a peritonsillar abscess he always endeavored to dissect the tonsil from its bed rather than attack the abscess through the pillar.

Dr. J. Lipschutz thought it was interesting to note the different attitudes of surgeons toward two similar conditions. Most surgeons think that the tonsils should not be removed in the presence of a peritonsillar abscess, and yet in cases of panophthalmitis, hardly anyone would advise incision of the eye with drainage, following it later with enucleation of the eyeball. Personally he would not care to remove tonsils in the presence of a peritonsillar abscess yet he wondered why the same did not hold true in panophthalmitis.

Dr. Norval H. Pierce said that it was very difficult to aspirate pus into the bronchi from the eye, and the control of hemorrhage in the eye socket was very much easier than control of tonsillar hemorrhage, so the conditions were entirely different. The dangers of a general anesthesia did not enter into the operation for

panophthalmia as they did in the operations on the throat, which made removal of the eye a very much simpler procedure.

Dr. J. Holinger thought it was a matter of individual opinion whether an operation of removing the tonsil in the presence of an abscess of which it is evidently the cause was considered surgical or nonsurgical. If one could show that after the incision of the abscess a second operation of removing the tonsil was avoided by this method, and that the experience of two men during a number of years had not produced any of the bad consequences that were supposed to be due to that special procedure, he felt that it should not be condemned. Neither Dr. Ostrom nor Dr. Holinger observed abscesses of the lungs or any cases of pneumonia following these operations, possibly because of good luck. The extirpation of the tonsil in the presence of an abscess is usually much easier than later on. Everyone had the experience of how difficult it was to dissect out a tonsil where there had been several peritonsillar abscesses in the course of preceding years. Large masses of scar tissue were found radiating in all directions and all the tissues were much tougher, so that it was practically impossible to pull the tonsil out of the niche in order to dissect it. The period of recovery, too, is much shorter in the first instance. Some cases of peritonsillar abscesses lasted six weeks and even three months, but with his procedure the patient was well within a week. He doubted if one was justified in calling such a procedure unsurgical. It was peculiar that those gentlemen who tried this operation were in favor of it and those who condemn it admit that they never tried it.

THE CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of May 3, 1920.

THE PRESIDENT, DR. ALFRED LEWY, IN THE CHAIR.

Cholesteatoma.

Dr. J. Holinger reported on the case shown at the April meeting. After showing the patient in the meeting of April the masses were removed and the perforations in the membrane and in the roof of the canal closed temporarily. About three weeks later it opened again, and through the roof of the canal a fourth lump of cholesteatomatous matter was discharged, which was offered for inspection. The opening at this time was closed except in two or three moist spots. The patient's hearing was improved and he felt much better.

The discharge of cholesteatomatous globules was described more than twenty-five years ago, but that in the course of three months four masses of the size of a pea should be discharged was unusual. Furthermore, that the opening in the membrane and in the roof of the external canal closed teaches that we may have an accumulation of cholesteatomatous masses in the middle ear and antrum in the presence of a practically normal membrane and canal.

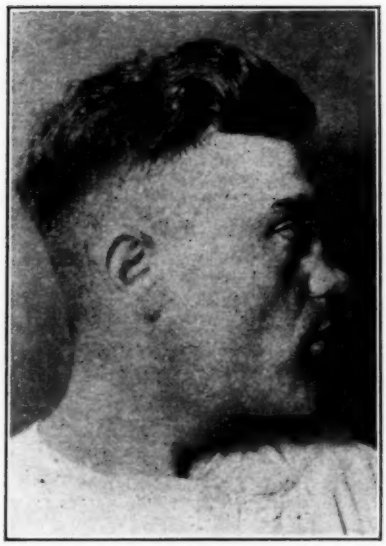
Fibroma of the Antrum.

Dr. Austin A. Hayden showed a patient with a tumor originating in the antrum, filling the entire side of the nose and extending out into the cheek, which had been growing for ten years. The patient had received five doses of radium in the past month, with considerable reduction of the growth. The size of the tumor and the history were unusual. The growth was a fibroma, probably of the mixed tumor type, but the pathologic report had not yet been received. Radiograms showed destruction of practically all of the teeth in the right upper jaw. The tumor was about the consistency of an overripe orange. The Wassermann reaction was negative and there was no history of syphilitic infection. (See photographs of case.)

Saddle Nose.

Dr. Hayden also presented a patient whom he had operated for a saddle nose, which resulted from trauma from a horse kick a year and a half previously, and showed a photograph of the patient before operation. A strip of cartilage one and one-fourth inches long had been taken from the nose of another patient, who was having a submucous operation done at the same time. Careful tests (blood-Wassermann) showed conditions to be favorable for the use of the cartilage. The operation was performed under local anesthesia, 1/10 of 1 per cent cocain being used subcutaneously. The incision was made well above the mucocutaneous junction. The periosteum and skin were freely elevated with an ordinary straight submucous elevator. Two implants were made, a long one of cartilage and a shorter one of bone, the bone being placed below. The cosmetic result was excellent. (See photographs.)

Tumor of Face.



Saddle Nose—Before Operation.

Saddle Nose—After Operation.

Paper: "The Whys and Wherefores of a Vestibular Examination."*

By Dr. Lewis Fisher, Philadelphia.

Dr. Fisher thought that the time had passed when it was necessary to do any preaching about the value of an examination of the entire inner ear. It is pretty well admitted that a complete ear examination is very useful in many ways. He presented three case histories and used them as a text for his remarks.

(Dr. Fisher than gave a lantern slide demonstration of charts and photographs.)

These presented a schematic outline of the nerve pathways in relation with the inner ear, as far as known to date. Taking up the three case histories in detail he pointed out the abnormal or absent reactions found on examination of these patients. With the conception of the nerve pathways of the vestibular apparatus as demonstrated on the schematic charts, these abnormal responses suggested certain locations of the lesions in these cases. Subsequent autopsies verified these suggestions.)

DISCUSSION.

Dr. Thor Rothstein said that when the speaker admitted that there were many cases which he did not know what to make of, it made him feel at home, because the same thing had happened to him. He was familiar with Dr. Fisher's work and the published work in connection with Jones, and that book is so lucid and clear that anyone who reads it must do so with awakened interest in all the diseases of the internal ear. It had been a great pleasure to him to hear Dr. Fisher. It was by this means that a very good working basis would be furnished that would give a foundation upon which one might try to make a diagnosis with an insight that had not existed before. The vestibular tracts had always been very mysterious to him, but there existed in these sufficient tracts to make it possible that Dr. Fisher was correct in his idea of these different parts. It was, of course, necessary that much work must be done, and the testing of the internal ear and the whole apparatus could not very well be done by anyone except an otologist, because it is necessary to have great experience to be able to judge of the results. For that reason there should be close cooperation between the otologist and the neurologist in these cases. Dr. Fisher had very clearly pointed out the difficulties; the difficulties in the central and peripheral portions, and the difficulties with a tumor that lies in the cerebellar pontine angle. Dr. Rothstein had observed many of these difficulties and thought there was always room for doubt as to whether it was possible to draw certain conclusions from an examination. For instance, in cases where there are severe attacks of vertigo in migraine, and also in cases where the vertigo is not so violent but remains for a longer time, there may be spontaneous past pointing and absence of the turning past pointing. Although the past pointing is very frequent in these cases, it is irregular and limited many times to jerkiness, and he wished Dr. Fisher would explain this. There were also other points for the man who is not very well versed in this examination. For instance, in the cases where there are ataxias; in these cases there

may be the phenomenon of past pointing, but in the few Dr. Rothstein had seen it had often been irregula rand might be the cause of mistakes. So there were many different things in examining these very dfiiicult cases which make it necessary that the man who makes the examinations must have great opportunity to observe what happens in these cases. In an individual that has been considered apparently normal, in whom it has been impossible to demonstrate any kind of disease, the accuracy with which the tests are made is not very good, not even in those who have lost past pointing.

In closing, Dr. Rothstein thanked Dr. Fisher for what had been to him a very impressive and interesting exhibition.

Dr. Hugh T. Patrick said he did not know very much about the matter, but the thing that pleased him most of all in Dr. Fisher's presentation was his extreme modesty, and the very small claims he made for the value of the examinations. He thought that in the past the value of the tests frequently had been overestimated. He thought Dr. Fisher would agree with him that it takes an enthusiast to make progress. But the enthusiasts who lead generally go a little too far and have to be checked up a bit, and he thought this applied to the men who had given us this very great addition to medical science. The moral was that these examinations should be made with extreme care and exactitude, and he believed that no man should allow himself to draw conclusions unless he had done enough of the work to be a real examiner and not a pseudo.

Dr. Patrick had an impression that a good many of the conclusions where the lesions involved the central nervous system have been based on tumor cases. Intracranial tumor cases are the worst cases in the world as a basis for refined localization. They have symptoms due to increase of intracranial pressure and to the peculiar way in which this pressure is exerted. This accounts for some of the symptoms at a distance. They are poorly adapted for localization of function, identification of paths or anything of that sort. Dr. Patrick thought the thing that was needed was a series of thrombotic cases carefully worked out. Such a series carefully observed and then carefully worked out anatomically would be a reasonable basis for anatomic and physiologic conclusions. In other words, the lesions which approximate what Horsley, Beaver and Clark did and others are trying to do—this is, make a limited lesion in the brain, will be those on which one might base a dogma of localization; not the cases of neoplasm or other things which cause increase of intracranial pressure.

Dr. J. Gordon Wilson said the members of the society were delighted to have Dr. Fisher with them and had listened with pleasure and no little instruction to his presentation of this important subject. The School of Pennsylvania had focused the attention of neurologists on the possibilities that can come from a careful investigation of reactions from the ear. They have shown admirable team work in their clinical studies, and to these have been added in many cases the pathologic postmortem examinations. While one can have nothing but praise for such careful reports, one may well be excused from hastily accepting some of the deductions based on them.

There are some generally accepted principles which are of aid in interpreting vestibular reactions:

1. If labyrinthine stimulation produce deviation but no nystagmus, then there is a lesion of the cerebral hemisphere near the posterior end of the temporal lobe on the side of the slow deviation. This was shown experimentally by Dr. Pike and Dr. Wilson and reported in a contribution to the International Congress of Medicine in 1913, and has been confirmed by clinical cases with postmortem findings, one of which was reported by Dr. Fisher in his address.

2. Nystagmus, vertigo and pointing produced by labyrinth stimulation are exaggerated in cerebral disease, probably due to loss of the controlling influence of the cerebral cortex. Here we have possibly the explanation of their exaggeration in neurasthenia.

To these the speaker added two other points which can be easily tested and which deserve consideration: (a) The threshold for nystagmus lies below that of vertigo, i. e., the motor reaction comes first. (b) The interpretation of a perverted eye reaction, for example, rotatory nystagmus following stimulation of the horizontal canal, must be interpreted with caution for the following among other reasons:

1. When nystagmus in animals deprived of one labyrinth is fading away the horizontal frequently gives place to a rotatory.
2. On stimulating the labyrinth with an electric current of energy far below that producing typical nystagmus, a rotatory movement is first produced.

Too much stress is apt to be laid on pointing errors. They are frequently irregular and an average of repeated examinations are required to arrive at accurate data. As Dr. Fisher said, pointing is a cerebral act, and probably the cerebellum comes into play to influence the accuracy of the movement. That does not necessarily require a special cerebellar center for each particular movement as has been urged. A considerable number of presumably normal individuals do not give a typical pointing either before or after turning, and a typical error may only appear after repeated rotation trials. As a general rule, we may recognize that deviation of the arm is normally produced by rotation. Dr. Wilson urged that we do not lay undue stress on a diminution and be careful in our interpretation of its absence.

Further, he believed it a mistake to lay too much stress on the degree of postrotatory vertigo or even of its absence. Mygind found that in a series of normal individuals examined only 34 per cent had postrotatory vertigo and some of these only after repeated rotations.

In regard to the separation of the fibers from the horizontal and superior canals in the pons and medulla, we must recognize that Dr. Fisher claims only the possibility of such, supported by clinical observation and postmortem examinations. One may hope to have supporting microscopic findings. Dr. Fisher recognizes that so far no experimental or anatomic findings support this hypothesis, and readily acknowledged that it is not satisfactory to rest such an important point on variations or alterations of pressure in the brain. So far Dr. Wilson's results have been negative. Recently

he had an opportunity to examine repeatedly a case of probable interference with the circulation in the posterior inferior cerebellar artery such as Dr. Fisher had quoted. In this case the area involved was evidently smaller than in his. There was evidence of interference with the vestibular mechanism on the side of the lesion. There was absence of pointing error in caloric stimulation from both sets of canals on the side of the lesion, and while on rotation pointing irregularities following stimulation of the horizontal and of the perpendicular canals were present, these were too irregular to support any deductions with regard to separation of fibers. But it should be recognized that one positive finding is worth a dozen negative.

There was much of Dr. Fisher's paper with which one could agree, and the speaker admired his presentation of the work done. He had focused attention on a very important subject and shown how clinical cases are to be examined and may be explained, and how the otologist can be of assistanec to the neurologist.

Dr. George W. Hall was interested in the case referred to by Dr. Wilson. In the last issue of the Journal of the American Medical Association, he reported a case of occlusion of the posterior inferior cerebellar artery, in which the vestibular findings were worked out by Dr. Brawley. He did not know whether Dr. Fisher had seen that article. The trouble with the posterior cerebellar artery occlusion is that the symptoms are not the same in all the cases. In some cases the lesion may be sufficient to produce pressure on the opposite side of the brain stem, as had happened in this case. The lesion was on the right side, but the man had disturbances on the left side of the brain stem as well, although they were slight. In the case referred to by Dr. Wilson the findings were very much more limited, and he believed the findings were more accurate than in the other case. He was interested in the vestibular findings because so far as he could find out Dr. Fisher's report on involvement of the cerebellar artery was the first on record in which the vestibular findings were recorded, and the case they were working on was the third in which the ear findings had been worked out with any degree of accuracy as a result of occlusion of the posterior cerebellar artery. That is the region which involves as a constant symptom the sensory portion of the fifth nerve. In every case he had been able to find in the literature up to that time, the fifth nerve was involved as the only constant symptom, but there might be other symptoms of involvement. In this case the localization was so limited that the only symptoms he had to go by were the anesthesia of the right side of the face and of the tongue, supplied by the sensory portion of the fifth. In addition to that, the man complained of dizziness; he had a constant tendency to walk to the right and vertigo when he arose to an upright position. The examination was not quite finished, but so far as Dr. Hall could see, the lesion was so extremely localized that the report should be of value as an addition to this line of work.

Dr. I. Leon Meyers understood Dr. Fisher to distinguish two sets of fibers, one for vertigo and one for nystagmus. It might be well to consider in this connection that vertigo, as well as its related phenomena, namely, forced movements, conjugate deviation of the head and eyes, and nystagmus are in reality different mani-

festations of one and the same disturbance and are unquestionably due to an affection of one and the same set of fibers. What happens is that the patient, as a result of a disturbance in the vestibular system, becomes conscious of moving in a certain direction—e. g., to the right. He may then look to the right by turning his eyes in that direction, thus forming the slow component of nystagmus, or he may look to the right by turning his head as well as his eyes, exhibiting a conjugate deviation of the head and eyes, and in case the disturbance is very profound he may even fall to the right. Vertigo is a minor manifestation of this disturbance. It is merely the sensation of the motor phenomena which exhibit themselves in the nystagmus and its allied symptoms. This sensation may exhibit itself subjectively or objectively, the patient experiencing the sensation of himself moving, or of the objects around him moving.

Dr. Fisher was in accord with the findings of Dr. Wilson regarding the production of the quick component of nystagmus. Dr. Meyers witnessed some experiments at the University of Chicago in which the quick component was observed in rabbits after the entire cerebrum had been removed. Dr. Ivy performed these experiments. He also performed them on dogs, but Dr. Meyers did not see those. From personal observation, having performed a large number of cerebellar operations on animals, he was convinced that nystagmus is not cerebellar in origin. He had succeeded in removing the entire cerebellum without producing nystagmus. Barany's past pointing tests in cerebellar disease, spoken of by Dr. Fisher, he had tested a good many times, but Dr. Meyers was not able to speak as to their value at the present time.

Dr. Austin A. Hayden took occasion to pay his respects to the sincerity and honesty with which Dr. Fisher had proceeded in his investigations. He had had the pleasure of taking his course at the University of Pennsylvania. He wished to make a special plea to the profession and especially to the internists, neurologists and eye, ear, nose and throat men, that they routinely submit more material for neurootologic examination and investigation, following the exact plan worked out by Jones and Fisher. This received wide and just popularity in the ear, nose and throat sections of the various physical examination units of the Aviation Section of the Signal Corps during the late war. The need of a standardized technic—universally recognized and adopted by all men doing this work—is clearly apparent in the statistics quoted by Dr. Gordon Wilson. If this were in use, it would hardly be possible for 24 per cent of any considerable series of cases to show no vertigo.

In the series of 3,748 and over which he reported in detail, June, 1919 (Annals of Otol., Rhinol. and Laryngol.), the percentages were as follows:

Nystagmus.—After rotation to the right there was an aggregate of 82,231 seconds, or an average of 21.9 seconds each. After rotation to the left there was an aggregate of 82,373 seconds, or an average of 21.9 seconds each.

Past Pointing.—After rotation to the right, the right arm past pointed 7,201 times, averaging 1.89 times each, and the left arm past pointd 6,663 times, making an average of 1.77 times each. After rotation to the left, the right arm past pointed 8,029 times,

giving an average of 2.14 times each, and the left arm past pointed 7,083 times, averaging 1.88 times each.

Of the 3,748 applicants only 142 were rejected for abnormalities of the kinetic static sense—a little less than 5 per cent; of these, 110 failed in past pointing, 17 in nystagmus, and 7 in falling; 22 men were nauseated and 31 vomited. Disqualification on account of freak causes numbered only 692, or 18.4 per cent.

The common sources of error set forth in the original paper are always to be guarded against.

It was a great source of regret to the men who were doing this work, to Dr. Pierce first of all, and to himself and Dr. Lewy later on, that they were unable to submit all of the rejected men to the caloric tests. The individual findings of the semicircular canal or central nervous system would doubtless have been most interesting to work out.

Dr. Hayden thanked Dr. Fisher for his very interesting presentation of this comparatively new but extremely important subject. He hoped it would be productive of many farreaching results. The most important of these would be a stimulus to more frequent neurootologic examinations.

Dr. Norval H. Pierce expressed his appreciation of Dr. Fisher's having come such a great distance in response to the invitation of the society, and thought he voiced the sentiments of nearly every one as to the great practical value in our everyday work of the knowledge already at our disposal regarding the vestibular apparatus. Owing to the very complicated anatomic factors it would take years before it would be possible to finally place the action of the end organs on a sound foundation. The anatomic space involved in the nerve paths and ganglia is so limited and the centers and tracts so numerous that it is going to be very difficult to differentiate in many cases. For example, the inhibiting mechanism of the vestibular action is not understood. In examining the aviators they saw several cases where profound nystagmus was produced by rotation and the individuals vomited. The very moment the vomiting occurred the nystagmus ceased. Therefore, there is an inhibiting mechanism in this region which inhibits nystagmus, notwithstanding the fact that the lymph is still exerting its action on the end organ. He did not believe this was due to pressure within the endolymph, and secondarily on the perilymph induced by increased abdominal pressure in the act of vomiting. This increases the pressure in the cerebrospinal fluid, it is true, but he had gone over some experiments to eliminate this as a cause of inhibition of nystagmus. It will take a long time and a great many autopsies on cases which have been carefully and accurately studied clinically before the many problems are solved. The matter was not always as plain as the books would indicate. Still, no one can question the great practical value of functional tests of the static apparatus in our everyday clinical work.

Dr. Alfred Lewy said that Dr. Fisher made a reference to a partial paralysis of the soft palate in its failure to lift at its base. Dr. Lewy had seen two instances of this kind in lesions that were supposed to involve the glossopharyngeal nerve. Instead of drawing up quickly the mucous membrane of the posterior wall of the pharynx seemed to draw over to the side, much as the face does in

facial paralysis. He asked if Dr. Fisher had ever noticed this condition in angle tumors, especially those that extend down and involve the foramen.

Dr. Lewis Fisher (closing) said that unfortunately there has been a tendency in the last few years to refer to a large phase of this work by the simple term of "past pointing." One is continually asked, "Have you done the past pointing tests; did the patient show past pointing or did he not show past pointing?" This attempt to sum up so comprehensive an examination in so short a phrase is probably due to a haziness of conception of the subject. Any attempt to sum up a vestibular examination in any such manner is like attempting to sum up a neurologic examination in the simple term of "knee jerk." The pointing tests per se, as a matter of fact, do not mean so much. Thus, from the standpoint of clinical diagnosis spontaneous past pointing scarcely means anything at all. Some individuals are naturally inaccurate, and one must not attach too much significanec to the fact that the patient misses the examiner's finger. Furthermore, many patients, even if they did have genuine pathologic spontaneous past pointing as a result of some lesion of the central nervous system, may after a while learn to compensate, and when examined such patients may find the finger accurately.

However, past pointing after stimulation is of greater significance, but after all it is simply an objective expression of the vertigo that is induced and may be easily affected or modified by many conditions, pathologic or otherwise. It is the presence or absence or the character of the induced vertigo that is of importance, and not so much its resulting past pointing. A thing to remember is that "past pointing" after stimulation is really normal pointing for the individual at that time. The stimulation induces a subjective sense of rotation—that is, though sitting absolutely still in a chair he thinks he is turning away from any point he has touched. When he thinks he is turning to the left of the examiner's finger he brings the arm down to the right, or "past points" to the right, and vice versa. To the examiner this appears as "past pointing," but to the patient it is a correct physiologic pointing.

The vertigo that is induced is also a normal physiologic function. It is simply the recording within the brain of the motion sensing that has occurred as a result of the lymph movement within the semicircular canals. The physiology of this method of motion sensing is as follows: When the head is turned to the right, with the head in an upright position, the lymph within the horizontal semicircular canals lags and moves relatively to the left. The individual knows that he has turned to the right; as a result of innumerable repetitions of this same act the brain learns to interpret that whenever the lymph has moved to the left it indicates a body movement to the right and vice versa. Therefore, when the patient is first turned in the chair to the left he says when questioned that he is going to the left; that is not called vertigo, because his statement is in accord with fact. If the turning is repeated fairly rapidly in a smoothly revolving turning chair we reach a point when the lymph within the horizontal canals catches up with the speed of the body and is relatively not moving at all. The patient then will say, if questioned, that he is not moving at all, while, as a

matter of fact, he is being rotated rapidly. If then the chair be suddenly stopped the lymph continues to move to the left as the result of the momentum imparted to it by the turning. This lymph movement to the left is immediately interpreted by the brain as body movement to the right, and the patient will say that he is turning to the right when, as a matter of fact, he is sitting absolutely still. Here then we have an instance of information received by the brain along perfectly normal physiologic channels which is not in accord with fact. This "apparently wrong" information is called vertigo. These impulses, started within the ear, naturally traverse definite pathways to the cerebral cortex, where they are received and accurately interpreted.

The nystagmus or eye movement impulses have nothing in common with the vertigo impulses except that for a portion of their course the pathways for these two are within one bundle. While they both start within the ear, one has the eye muscle nuclei as its objective, whereas the other must have the cerebrum as its objective since it results invariably in a definite concise conscious idea. As a result of disease some individuals when stimulated manifest one without the other—something which could not occur if they were both one and the same thing.

Dr. Wilson mentioned that he had never come across a case which would suggest to him that the fibers from the vertical canals have a different course than those from the horizontal canals. Dr. Fisher stated that he had seen a case that very day in Dr. Norcross' office where with a perfectly normal ear no response could be obtained from the vertical semicircular canals, but a very good active response from the horizontal canal was obtained. Evidently the pathways from these two sets of canals must not be together throughout their entire course. When one is very careful with the technic of the douching so that one set of canals alone is stimulated, a number of such instances will be encountered. The observation of Dr. Wilson that in cases of supposed occlusion of the cerebellar arteries one cannot elicit past pointing is in conformity with Dr. Fisher's experience. In these patients there is usually an involvement through a circulatory disturbance of one or more of the cerebellar peduncles. These peduncles carry the fibers concerned in vertigo and past pointing and are probably interfered with by the lesion. The fibers for nystagmus, however, are not interfered with in these cases, further suggesting that these eye movement fibers probably do not go to the cerebellum.

As for the rabbits having no quick component, Dr. Fisher said that while it may be true of rabbits, he did not know how true it was of human beings.

Clinically cerebellar lesions show spontaneous nystagmus. This, however, need not necessarily indicate that such a phenomenon is directly controlled by the cerebellum. The spontaneous nystagmus may be produced in these cases by pressure against the brain stem. Dr. Fisher believed that a lesion limited to the cerebellum and exerting no influence upon the brain stem would probably have no spontaneous nystagmus. From a practical standpoint, however, it matters very little, since most cerebellar lesions do affect the brain stem directly or indirectly and do exhibit spontaneous nystagmus.

Replying to Dr. Pierce, Dr. Fisher said that while it was true that we did not know everything about the vestibular mechanism, and probably never would, there is no reason for being discouraged. There is enough known for a good working basis. No doubt some of the present day ideas will eventually have to be modified, but, on the other hand, conclusions in so many cases that have been studied with the present conception of the vestibular mechanism have been vertified by autopsy or operation that one cannot help but be impressed that we have even today a fairly good working formula. Under no circumstances must it be understood that this type of examination is intended for general neurologic diagnosis. The cases discussed simply illustrated how helpful such an examination could be to the neurologist.

Replying to Dr. Lewy's remarks about involvement of the soft palate, Dr. Fisher stated that he had mentioned this simply as something which at times might be helpful in differentiating between a central and peripheral lesion.

Dr. Fisher thanked the society for the courteous reception extended him.

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